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SURGERY

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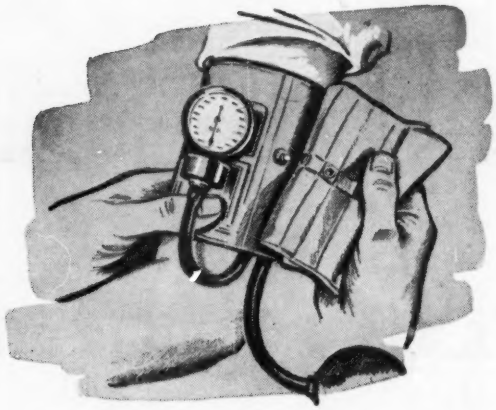

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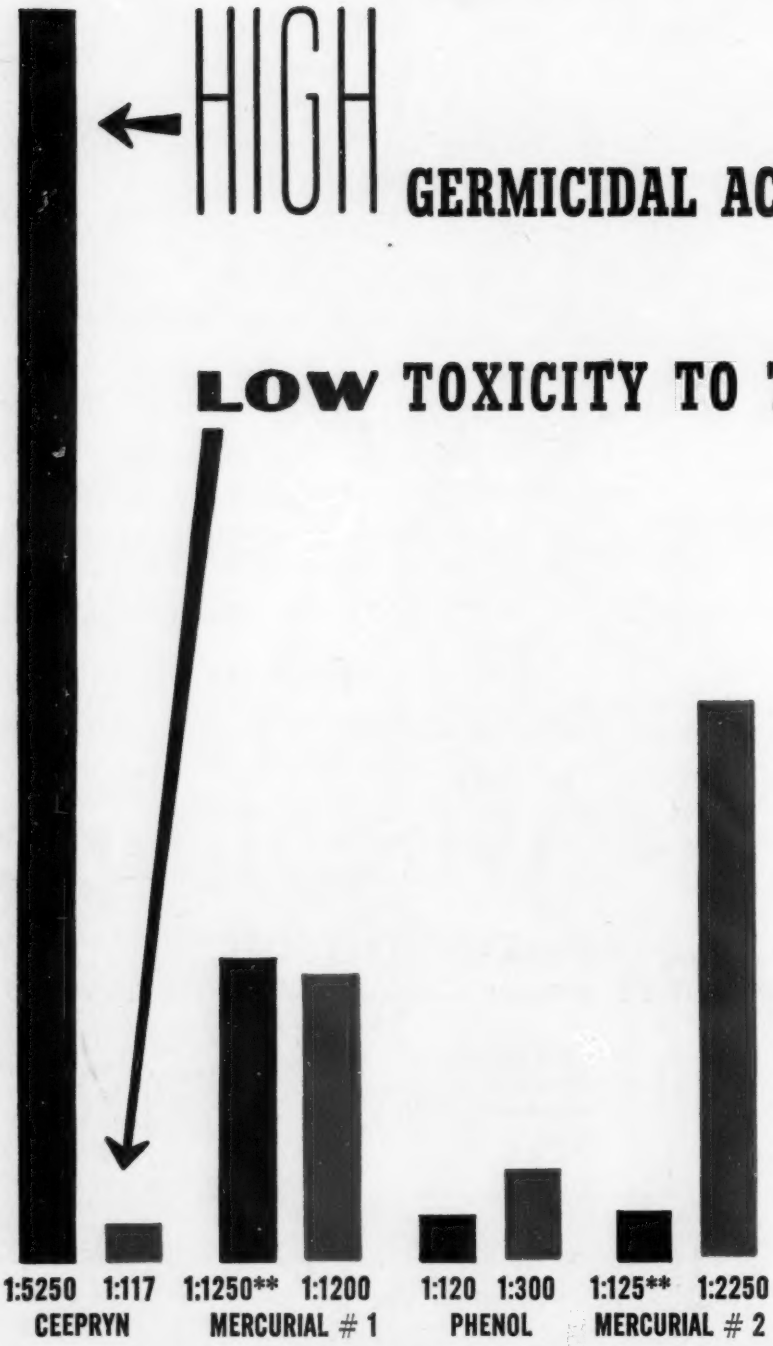

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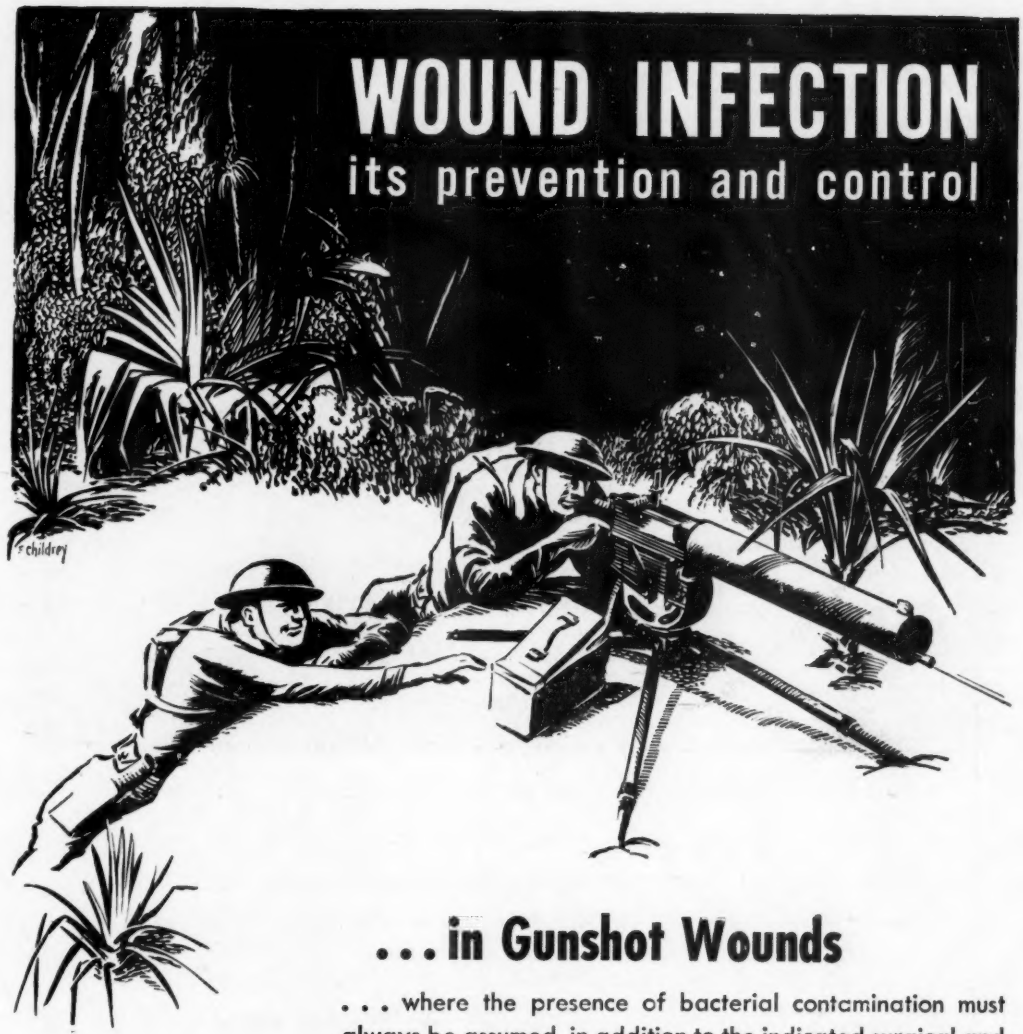

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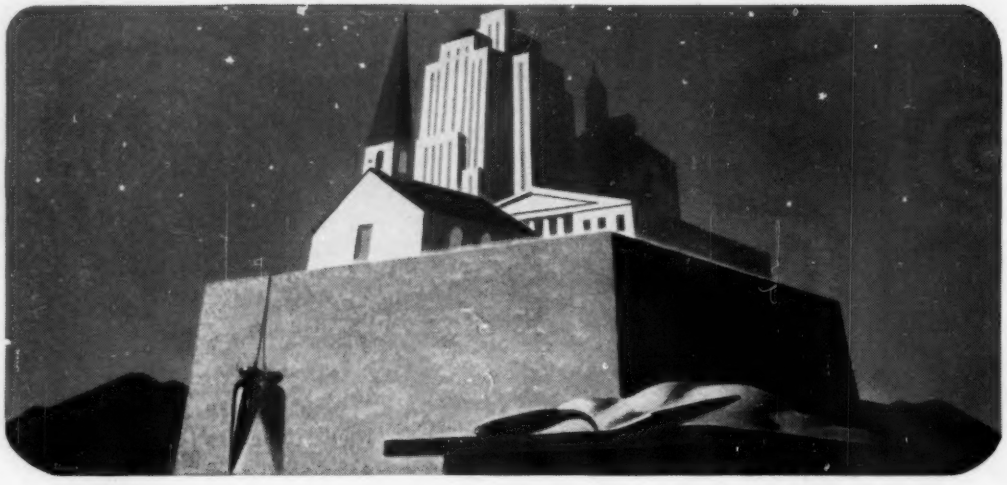

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Original Communications

SYMPATHECTOMY IN THE TREATMENT OF PERIPHERAL VASCULAR DISEASE

HARRIS B. SHUMACKER, JR., M.D., BALTIMORE, MD.

(From the Department of Surgery, The Johns Hopkins University School of Medicine)

DISORDERS of the peripheral circulation offer a particular challenge to the medical profession because of their common occurrence and their disabling character. Their treatment, largely neglected in the past, has been the subject of intensive study in recent years. Although sympathectomy is still a controversial issue, there is no doubt in my mind that it is one of the most valuable aids in the treatment of peripheral vascular disease. It is my purpose to discuss the importance of the study of cases in an attempt to evaluate the possible benefit which may be derived from sympathectomy, to describe the technique employed in sympathetic denervation of the upper end of the lower extremities, and to present the results of such operations in a series of cases which I have studied. It is not within the province of this communication to discuss the diagnosis of peripheral vascular disease. As Homans[1] has pointed out in his excellent monograph, the sorting out of the various disorders of the peripheral circulation is not a particularly difficult task and no elaborate diagnostic equipment is necessary.

THE STUDY OF CASES

Sympathectomy may be helpful in three ways: It may increase circulation through the elimination of vascular spasm; it may increase the warmth of the affected part by causing cessation of sweating and reduction of heat loss; and it may give relief from pain. Before a patient is subjected to this operation it should be determined whether he will be likely to benefit by it and to what extent. The method employed in this determination is best illustrated by a study of patients with arterial circulatory deficiency, the largest group to whom sympathectomy is applicable. Diminution in peripheral arterial circulation may be due to occlusion of vessels, to vasospasm, or to a combination of the two.

Received for publication, April 27, 1942.

It is essential to determine in which category any given case belongs. Blood flow cannot be restored through obliterated arteries, but vascular spasm can be effectively released. From history and ordinary examination, from the presence or absence of the major pulsations, from the character of the arteries on palpation and on x-ray examination, from the color changes in the extremity with alteration of position and after release of a constricting tourniquet, from the stability or lability of warmth of the affected part, much can be learned as to the degree to which vascular spasm contributes to the arterial circulatory deficiency. It is, however, important to measure the degree of vasospasm as accurately as possible.

The method employed is observation of the skin temperature of the affected part before and during inhibition of sympathetic vasoconstrictor impulses. Observations of thermal changes may be supplemented by oscillometry, various types of digital plethysmography, or plethysmographic study of muscle blood flow. The subject is first placed with the limbs exposed in a cool room, preferably one constantly maintained at 20° C. The rate and degree of cooling are noted by frequent measurements with a thermocouple. The digits often cool to room temperature, and they may become even cooler if there is sufficient loss of heat by evaporation from an actively sweating extremity. This phenomenon is commoner in the vasospastic diseases but may also be noted in obliterative arterial disease. After the skin temperature has become stabilized, sympathetic vasoconstrictor impulses may be eliminated in a number of ways. A simple and useful test is the thorough warming of the body, which may be done by a hot air cabinet,[2] by wrapping in blankets and a rubber sheet[3] or by the use of electric pads, and by diathermy over the lumbosacral area.[4] A similarly useful test is the immersion of the other extremities in warm water.[5] I occasionally employ such body-warming tests, particularly in order to study vasospasm both in upper and lower extremities at the same time. If good vasodilatation does not occur it is my feeling that some other test should be used as a supplementary check. Spinal anesthesia may be used in good-risk patients who are being hospitalized[6, 7] and is especially valuable when both lower extremities must be investigated. General anesthesia can also be used,[6] but is ordinarily not advisable unless anesthesia is necessary for some other purpose.

A simple and effective test which can be carried out without special equipment and on ambulatory patients is the study of thermal changes in the distribution of a peripheral nerve which has been anesthetized with procaine.[8]. I frequently employ posterior tibial nerve block for study of the feet and ulnar nerve block for study of the hands. Peripheral nerve block should not be carried out if there is infection or gangrene in the region where the injection must be made. The direct infiltration of the sympathetic nerves with procaine[9] is in many respects the ideal test, for it has the advantage over all the other methods

in permitting the study of the effect on pain of interruption of the sympathetic nerves. For this reason it should be used, in my opinion, whenever pain is present. One can study not only the effect on resting pain but also on intermittent claudication. The technique of injection is not difficult; it is not a painful procedure, it may be carried out on ambulatory patients, and if small doses of 0.5 per cent procaine with adrenalin are used and care is taken to avoid injection into the blood stream, pleural sac, and subarachnoid space, it is a safe procedure.[9] The accuracy of the injection of the upper dorsal sympathetics is attested by the presence of a Horner's syndrome. If doubt exists as to the accuracy of lumbar sympathetic injection, the matter can be checked by cutaneous resistance studies.[10]

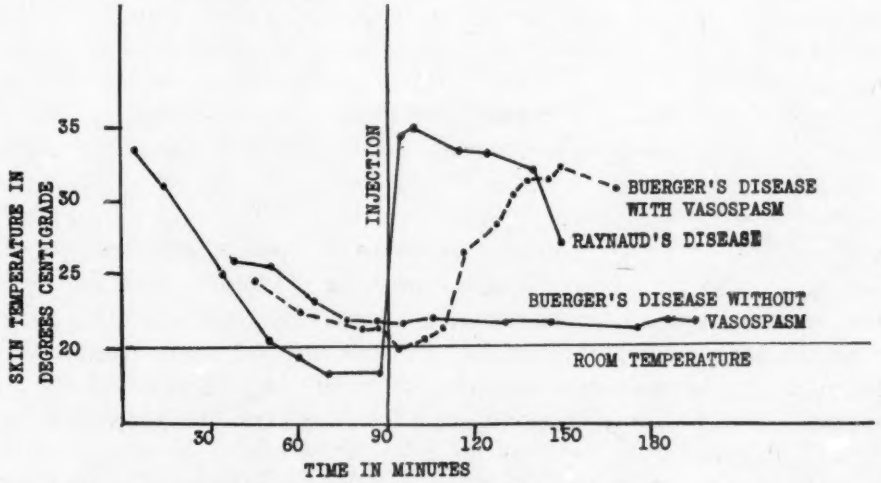

Fig. 1.—Skin temperature changes in a single digit before and after inhibition of vasoconstrictor impulses by sympathetic or peripheral nerve block in a case of Raynaud's disease and in two cases of Buerger's disease.

If more than one extremity is affected the element of vasospasm should be studied in the extremity with the most severe damage. One can assume then that the capacity of the vascular bed in the other extremities to dilate is somewhat better than or at least as good as in the extremity studied. Observation of the skin temperature in the contralateral limb serves as a control during study of thermal changes in the limb being investigated during inhibition of vasoconstrictor impulses. In Fig. 1 are charted examples of thermal changes after sympathetic or peripheral nerve block in cases of pure vasospastic disease, obliterative arterial disease with considerable vasospasm, and obliterative disease with little or no vasospasm. Misleading information may be obtained if the skin temperature of only a single digit is followed as was commonly done by the early workers. Fig. 2 illustrates that rise of temperature may be negligible in one digit but good in others.

Essentially the same sort of study is carried out in other types of peripheral vascular disease in which sympathectomy is under consideration. In cases of causalgia one is particularly interested in the

question of relief of pain. In postphlebitic states one should try to ascertain whether there exists demonstrable evidence of vasospasm in the affected extremity. Sometimes this is evident from skin temperature studies. Sometimes there is hyperhydrosis. In some instances I

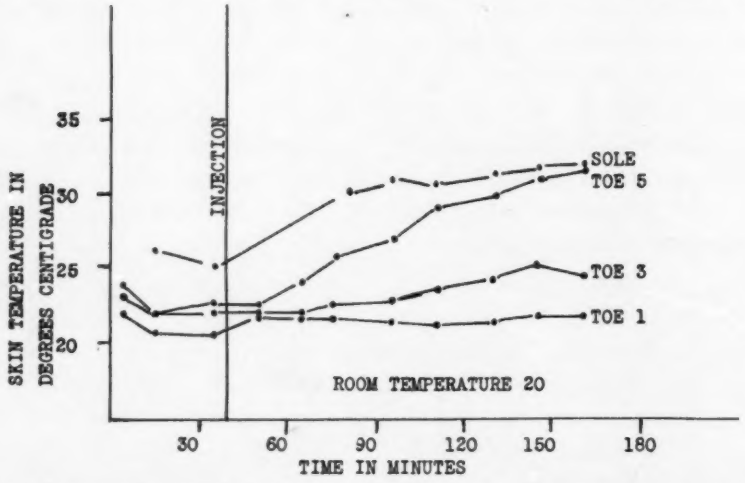

Fig. 2.—Skin temperature changes in a patient with Buerger's disease before and after posterior tibial nerve block. The variation in the response in different toes is illustrated.

have found an area of abnormally low cutaneous resistance and sometimes an area of hyperhydrosis in the region above the internal malleolus where pigmentation and ulceration so frequently occur, an observation which may be indicative of a localized region of vasospasm.

THE TECHNIQUE OF SYMPATHECTOMY

A preganglionic type of operation is employed for dorsal as well as lumbar sympathetic denervation. Regardless of whether those are correct who feel that the chief advantage of a preganglionic operation is in a lesser tendency toward subsequent development of adrenalin sensitivity,[11] this operation seems to give better results than a postganglionic operation. For lumbar sympathectomy I use with slight modification the muscle-splitting extraperitoneal operation which is commonly employed.[12,13] This approach is easier, permits a more adequate exposure of the entire lumbar ganglionated chain, and carries with it far less risk than the transperitoneal operation.

Spinal anesthesia is used as a routine procedure unless specifically contraindicated. The patient is placed on his back with the side to be operated upon slightly elevated by a sandbag under the corresponding hip. The skin incision extends from the tip of the twelfth rib to a point about midway between the umbilicus and the anterior superior spine of the ilium (Fig. 3). The external oblique muscle and fascia are divided in the line of the fibers and dissected back so as to expose adequately the internal oblique muscle; this is then divided in the direction of its fibers, the incision being placed so as to point to the

second lumbar vertebra or about three inches above the umbilicus. The incision is carried as far as the border of the rectus sheath. Care is taken to avoid injury to the underlying intercostal nerve. The transversalis muscle and fascia are similarly divided. Blunt dissection with the fingers is now carried down in the extraperitoneal space until

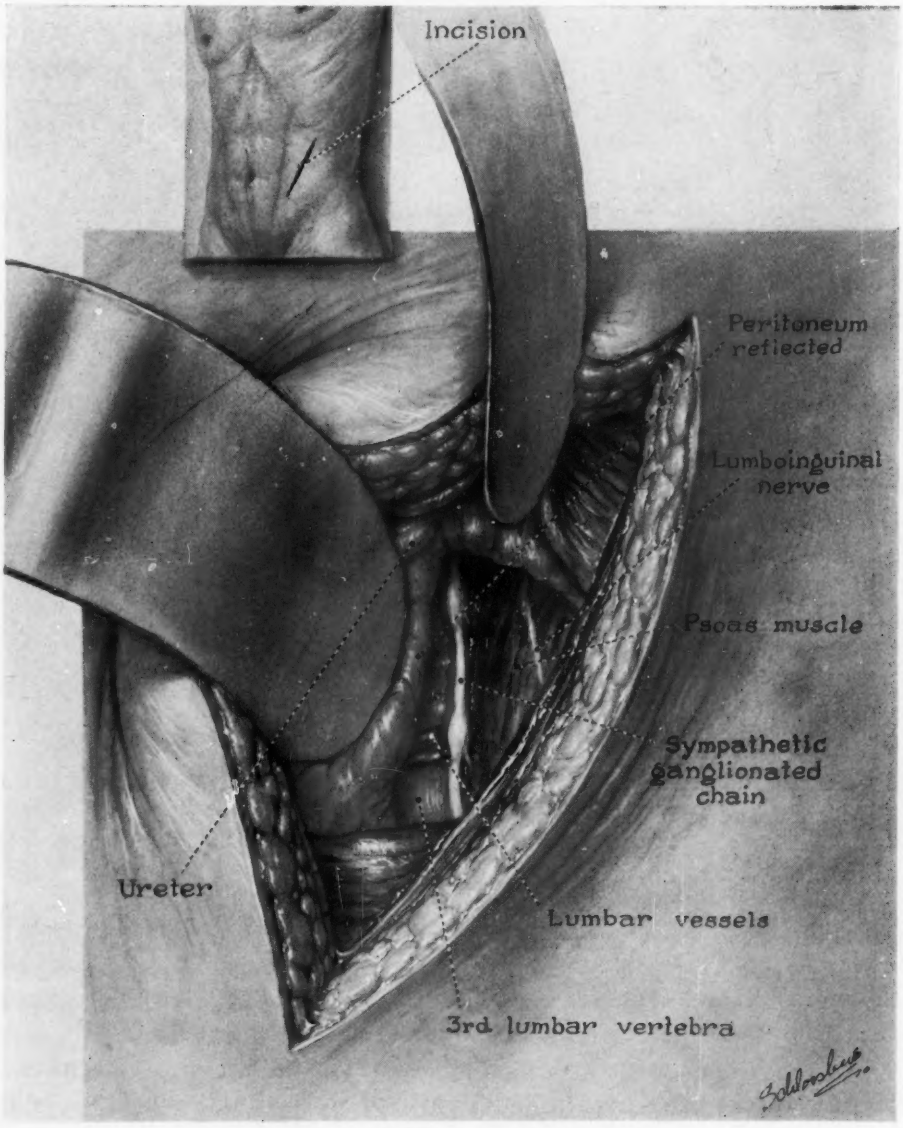

Fig. 3.—Lumbar sympathectomy.

the iliopsoas muscle is felt, then anteriorly over the muscle until the fingers palpate the body of the vertebra. One can readily identify the ganglionated sympathetic chain with the finger tips and can then proceed to dissect gently up and down along the course of the chain. A broad Deaver retractor is introduced and retracted mesially, ex-

posing the vertebrae, the aorta or vena cava, and the sympathetic chain. The ureter lies in the extraperitoneal fatty tissue, which is held out of the field by the retractor. The lumbo-inguinal nerve is seen lying on the iliopsoas muscle and should not be injured. A small Deaver retractor is placed in the upper or lower end of the wound as needed to

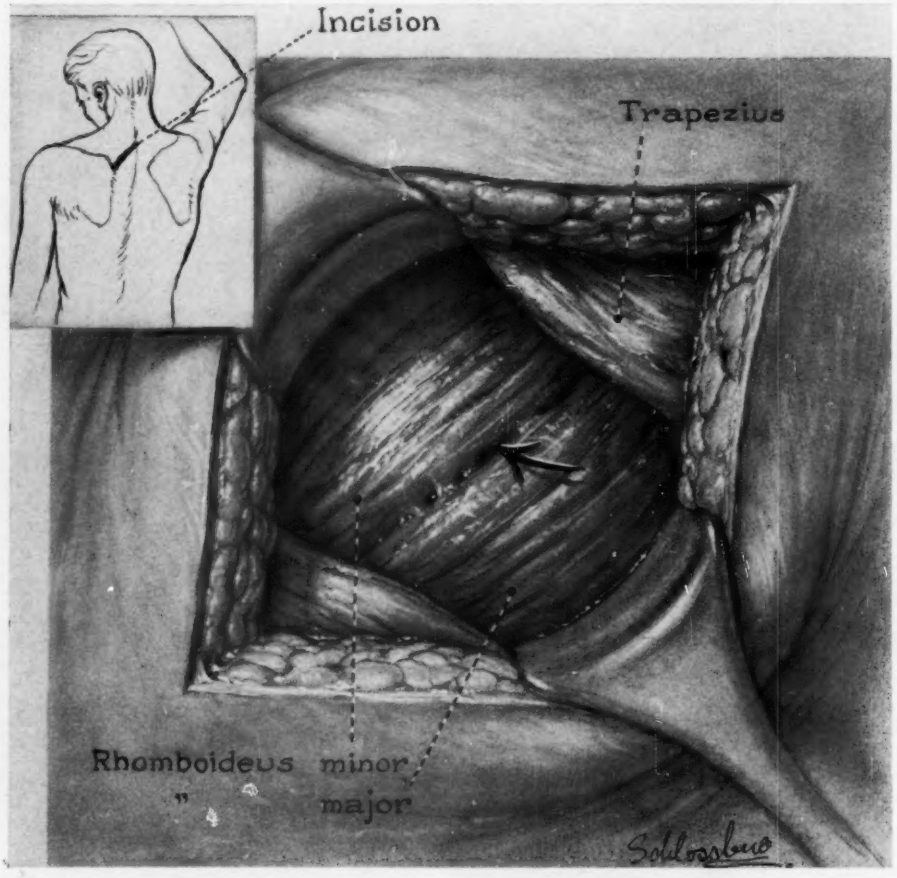

Fig. 4A.—Dorsal sympathectomy, showing position of the patient and line of incision. The trapezius has been incised and the arrow indicates point of separation of the rhomboids.

bring the upper or lower ganglia into view. A silk traction suture slipped around the chain by means of an aneurysm needle aids in dissection. The ganglia are identified. The fourth lies proximal to the promontory of the sacrum. The rami of the third and fourth ganglia generally point caudally and those of the second point cephalad. In females the first, second, and third ganglia and the intervening chain are resected. In males the second and third or the second, third, and fourth are resected, since removal of the first ganglion interferes with ejaculation. Ordinarily no bleeding occurs. The transversalis, the internal, and the external oblique muscles and fascia are closed with interrupted sutures of fine silk, and the skin is closed with interrupted

subcuticular sutures of fine silk. The operation can usually be completed in twenty to forty minutes.

The operation used for denervation of the upper extremities is essentially the upper dorsal sympathectomy proposed by Smithwick.[11] It is the same type of operation which Telford performs through an anterior incision.[14] A modified muscle-splitting posterior approach is used (Fig. 4). The patient is placed prone on the table with a sandbag

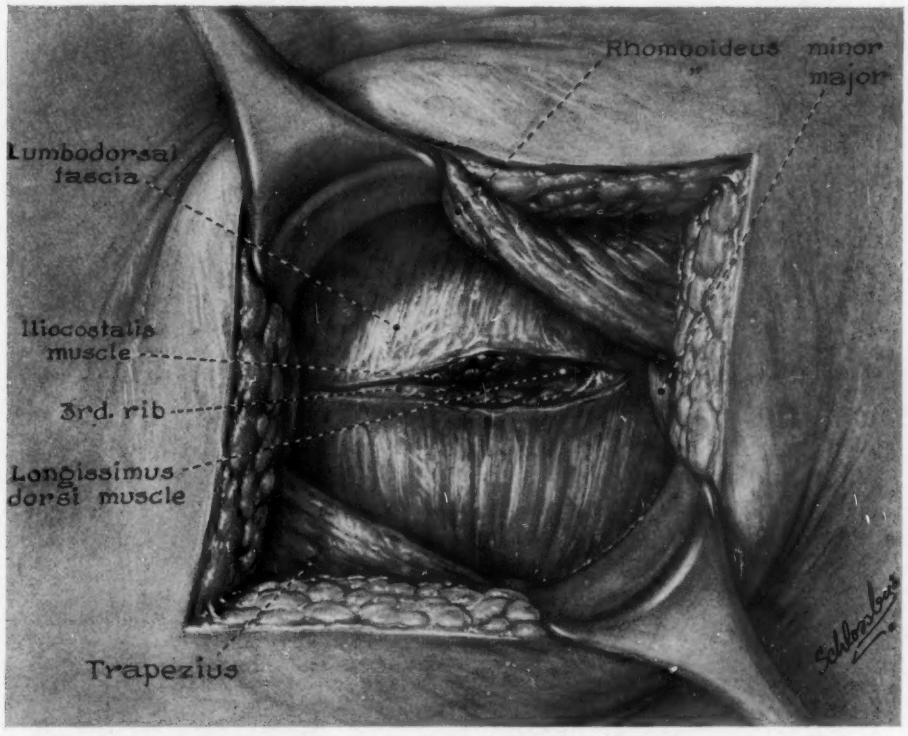

Fig. 4*B*.—Dorsal sympathectomy with incision through lumbodorsal fascia.

under the sternum, one arm lying alongside the trunk, the other forward along the head. The patient is anesthetized with sodium pentothal given intravenously. In my opinion this anesthetic is far superior to inhalation anesthetic with or without an intratracheal catheter. An incision 7 or 8 cm. long is made from the first thoracic spinous process to the spine of the scapula. The trapezius muscle is incised in the line of its fibers, thus exposing the rhomboid muscles, which are divided in the line of the fibers. The ribs are identified and an incision is made through the lumbodorsal fascia over the third rib. The mesial portion of this incision is continued through the iliocostalis cervicis. Sometimes a few of the lateral fibers of the longissimus dorsi are divided. The periosteum of the rib is incised and the inner 3 cm. of the rib are removed, care being taken to avoid opening the pleura. The pleura and the endothoracic fascia are pushed back gently from the second

and fourth ribs and from the vertebral body. The tip of the transverse process and the remaining head and neck of the rib are removed. The second and third intercostal nerves are identified and dissected free down to the dorsal root ganglia. Just beneath are the second and third sympathetic ganglia. A nerve hook is introduced between the anterior and posterior roots and by retraction these roots are brought into view. The dorsal root is divided and then the anterior root is divided either extradurally or intradurally. In many of my cases the division was

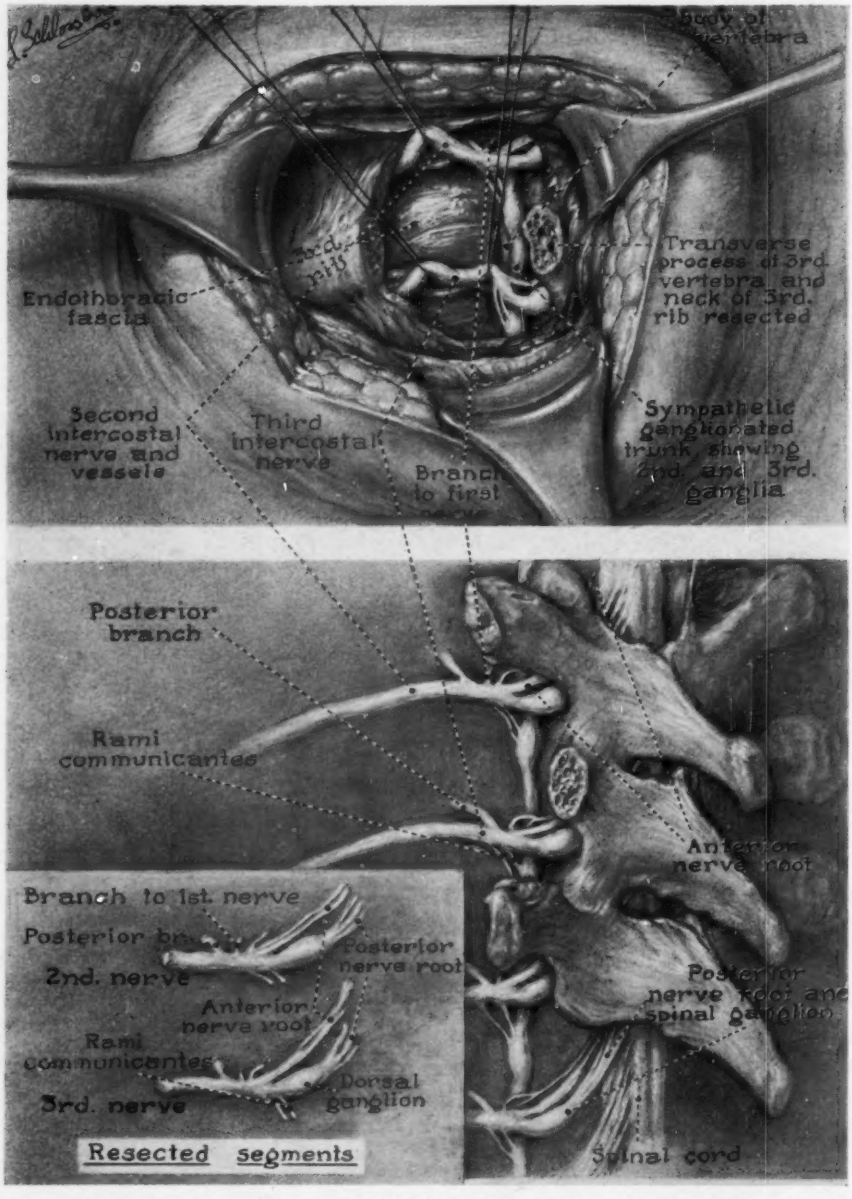

Fig. 4C.—Dorsal sympathectomy. The third rib and tip of transverse process have been resected; there is exposure of the intercostal nerves, anterior and posterior roots, and the sympathetic chain.

extradural; but Smithwick has suggested intradural division as an additional safeguard against regeneration, and I believe this procedure is preferable. The freed spinal ganglion is picked up and the rami to the sympathetic ganglion are divided. The intercostal nerve is now divided so that the excised specimen consists of the inner inch of the nerve with its ganglion and dorsal and anterior roots.

Fig. 4*D*.—Dorsal sympathectomy. The sympathetic chain is sutured to the muscle.

After this procedure has been carried out with both second and third nerves the sympathetic chain is divided caudad to the third ganglion and the freed chain and decentralized second and third ganglia are brought up into the wound and sutured to the muscle. A few interrupted sutures of fine silk approximate the iliocostalis muscle, the rhomboids, and the trapezius. The skin is closed with subcuticular sutures of silk. It is my custom to place in the extrapleural space before closure of the wound about 4 Gm. of sulfanilamide powder. Once in a while a pleural tear will occur, though this can usually be avoided by care in freeing and removing the rib and in stripping back the endothoracic fascia. When it occurs the lung is fully expanded by positive pressure before the wound is closed, and no harm results.

Patients recover quickly from both the lumbar and the dorsal operation. They are ordinarily able to sit up in a chair within a few days and may be subjected to a second operation in from four to seven days. Immediately after operation the denervated extremity is dry and warm to its maximum extent. Cutaneous resistance is high. A curious

phenomenon occurs on about the fourth, fifth, or sixth day. The hand or foot cools, cutaneous resistance falls somewhat, and sweating may even occur. The degree of this change is variable, and it is much more noticeable in the hands than in the feet. No entirely satisfactory explanation for this phenomenon has been found. It may be due to vasoconstrictor impulses arising in the decentralized sympathetics associated with degeneration of fibers. At any rate, within a few days the skin temperature again rises as does the cutaneous resistance, and sweating is impossible. This state of affairs continues indefinitely. Should degeneration occur at some future date, evidence of vasoconstriction would return. In the present series there has been no evidence of regeneration following either dorsal or lumbar sympathectomy. The area which has been denervated may be outlined by the Minor sweating test or by mapping out of the area of high resistance to the passage of a small electrical current.[15] The areas denervated by removal of the second and third lumbar ganglia, by removal of the first, second, and third lumbar ganglia, and by the dorsal sympathectomy are illustrated in Fig. 5.

RESULTS OF SYMPATHECTOMY

The present series consists of 83 sympathectomies performed upon 54 patients in the Johns Hopkins Hospital since November, 1939. Included are only those cases studied by me. The operations have been performed either by me or by the resident staff under my direction.

Results of Sympathectomy in Vasospastic Disease.—Twenty-six sympathectomies have been performed upon thirteen patients with vasospastic disease (Table I). Seventeen upper and nine lower extremities were denervated. Nine of the patients were females, four were males. Eight of them suffered from the common type of Raynaud's disease. Four additional patients had a Raynaud-like syndrome: One was associated with severe causalgia of the thumb following a bone felon; one was associated with prominent livedo reticularis; one was associated with purpura; two followed mild frost-bite. The result was excellent in all the patients save one. The exceptional patient (Case 2) was definitely improved, with cessation of attacks of vasospasm from emotional excitement but persistence of annoying sensitivity to cold. There has been no further alteration in his symptoms during the two years of observation. Six of the patients had varying degrees of scleroderma, in most instances very pronounced. In all of these there has been a considerable improvement in the scleroderma; the skin is softer and the digits are more mobile. Whenever digital ulcers or gangrenous patches were present they healed promptly and have not recurred. The patient in whom there was arthritis of the hands in addition to Raynaud's disease has not only been relieved of the attacks of vasospasm but has had considerable improvement in the arthritis. The girl with livedo reticularis has shown much improvement in this regard.

The young woman in whom there was purpura limited to the vasospastic lower limbs, and with negative hematologic studies and negative tourniquet test in the arms, showed little or no improvement in the purpura although this is well controlled with elastic stockings, and her coldness and numbness have disappeared. The patient with causalgia has had no further pain. One patient included in this group was a

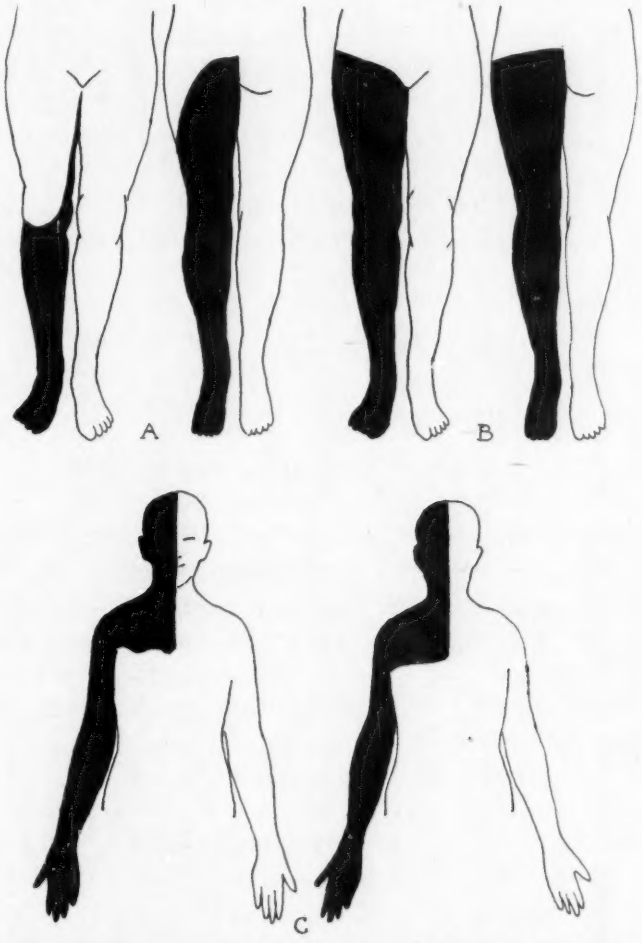

Fig. 5.—Showing the area denervated by removal of the second and third lumbar ganglia (*A*), by removal of the first, second, and third ganglia (*B*), and by dorsal sympathectomy (*C*), the areas inked in do not sweat on the Minor test and have abnormally high cutaneous resistance.

young man with elephantiasis in whom hyperhydrosis was so great that maceration of the skin occurred. It was felt hazardous to proceed with the plastic operation for elephantiasis until the hyperhydrosis had been corrected by sympathectomy.[16]

Results of Sympathectomy for Phlebitic Sequelae and Chronic Leg Ulcers.—Fourteen lower limbs have been sympathectomized in thirteen patients who suffered from phlebitic sequelae or chronic leg ulcers. Ten of the patients had had femoro-iliac thrombophlebitis; six of these

TABLE I

RESULTS OF SYMPATHECTOMY IN VASOSPASTIC DISEASE

CASE NUMBER	UNIT NUMBER	SEX	AGE	DIAGNOSIS	DURATION OF SYMPTOMS	SYMPATHECTOMY, DATE	RESULTS	PERIOD OF FOLLOW-UP
1	186027	F	44	Raynaud's disease with rapidly progressive scleroderma	2 yr.	Right dorsal, 1/15/40 Left dorsal, 1/25/40	Attacks of vasospasm ceased; scleroderma improved	2 yr.
2	115967	M	35	Raynaud's disease	4 yr.	Left dorsal, 3/11/40 Right dorsal, 4/8/40	Attacks of vasospasm from emotional excitement ceased; little improvement in coldness of hands on exposure to cold	2 yr.
3	198080	F	29	Raynaud's disease with scleroderma and digital ulcers	11 yr.	Left dorsal, 4/27/40 Right dorsal, 5/7/40	Attacks of vasospasm ceased; no further ulcers, scleroderma improved, fingers more mobile	2 yr.
4	114184	M	38	Raynaud's disease with scleroderma; loss of right finger 4 from gangrene. Contracture of right finger 5	5 yr.	Left dorsal, 4/9/40 Right dorsal, 8/25/41	Scleroderma greatly improved in left hand, moderately in right; attacks of vasospasm ceased	2 yr.
5	189243	M	33	Hyperhydrosis, elephantiasis, diffuse subcutaneous hemangioma	33 yr.	Right lumbar, 5/1/40	Sweating ceased; elephantiasis treated with good result by Homans' operation	2 yr.
6	202048	F	18	Severe vasospasm and livedo reticularis	2 yr.	Right lumbar, 7/25/40 Left lumbar, 8/1/40	No further coldness or numbness; livedo much improved.	21 mo.

7	186944	F	52	Raynaud-like syndrome; causalgia right thumb	3½ yr.	Right dorsal, 10/28/40	Pain entirely gone; hand remains warm	18 mo.
8	221998	M	35	Raynaud's disease with edema and rash on hands and feet, scleroderma ears	10 yr.	Left lumbar, 3/4/41 Right lumbar, 3/13/41 Right dorsal, 4/16/41 Left dorsal, 4/25/41	No further attacks of vasospasm, edema, or rash; scleroderma improved	1 yr.
9	215934	F	24	Vasospasm of the legs with purpura and edema; old history of frostbite	7 yr.	Right lumbar, 4/9/41 Left lumbar, 4/14/41	No further coldness or numbness and edema less; purpura continues but is controlled with elastic stocking	1 yr.
10	225486	F	60	Raynaud's disease with slight scleroderma; edema of hands, gangrenous ulcer on left index finger	5 mo.	Left dorsal, 4/12/41 Right dorsal, 4/21/41	Edema and attacks of vasospasm ceased, ulcer healed promptly; patient died 2 months later of Rickettsia-like fever	2 mo.
11	203914	F	19	Raynaud's disease with scleroderma	5 yr.	Right dorsal, 8/9/41 Left dorsal, 8/16/41	No further attacks of vasospasm; scleroderma improved	8 mo.
12	175179	F	42	Raynaud's disease; arthritis in hands	10 yr.	Right dorsal, 11/26/41 Left dorsal, 12/2/41	No further attacks of vasospasm; edema disappeared, mobility improved and arthritis considerably improved	5 mo.
13	108840	F	38	Post-frostbite vasospasm	3 yr.	Right lumbar, 3/20/42 Left lumbar, 3/24/42	Attacks of vasospasm and of burning pain ceased	1 mo.

TABLE II

RESULTS OF SYMPATHECTOMY FOR PHLEBITIC SEQUELAE AND CHRONIC LEG ULCERS

CASE NUMBER	UNIT NUMBER	SEX	AGE	DIAGNOSIS	DURATION OF SYMPTOMS	SYMPATHECTOMY, DATE	RESULT	PERIOD OF FOLLOW-UP
14	204612	F	52	Recurrent femoral thrombophlebitis, postphlebitic edema, pain and vasospasm	13 yr.	Left lumbar, 6/21/40	Discomfort less, edema slightly improved, no vasospasm; attacks of phlebitis continue but milder and of shorter duration	22 mo.
15	131399	M	56	Postphlebitic ulcer and edema	32 yr.	Left lumbar, 9/3/40; excision ulcer and skin graft 9/19/40	Ulcer healed rapidly with no further difficulty; doing hard labor	20 mo.
16	248107	F	37	Postphlebitic elephantoid state; chronic and recurrent femoro-iliac phlebitis	14 yr.	Right lumbar, 9/9/40	Only one brief recurrence of thrombophlebitis; leg much more comfortable but swelling persists	20 mo.
17	106934	M	38	Postphlebitic ulcer and edema	5 yr. ulcer 3 yr.	Left lumbar, 9/23/40	Had small pulmonary emboli, treated by iliac vein ligation. Ulcer healed. Small ulcer recurred 5 mo. later; healed with pressure bandage. Working	19 mo.
18	227848	M	32	Postphlebitic pain and edema	10 yr.	Left lumbar, 5/2/41	Discomfort gone, edema less; leads active life	1 yr.
19	238685	M	49	Postphlebitic ulcer	3 yr.	Left lumbar, 8/25/41	Ulcer healed, edema less	8 mo.

20	246266	M	30	Postphlebitic ulcer, edema and pain	6 yr.	Right lumbar, 11/22/41	Ulcer healed, pain diminished	5 mo.
21	246105	M	37	Postphlebitic ulcer and pain	4 yr.	Left lumbar, 11/25/41	Ulcer healed promptly, small pin point recurrence when started to work; controlled with pressure bandage. Pain gone.	5 mo.
22	245430	M	29	Postphlebitic edema, cutaneous and subcutaneous hyperesthesia	1 yr.	Left lumbar, 11/26/41	Pain and hyperesthesia gone, edema less	5 mo.
23	247544	M	27	Postphlebitic ulcer and edema	8 yr.	Left lumbar; excision ulcer, skin grafts, 12/3/41	Ulcer healed, edema less, discomfort less; working	5 mo.
24	221580	M	43	Post-traumatic (fracture) edema with painful ulcer	35 yr. ulcer 10 mo	Skin graft, 2/17/41 Right lumbar, 2/24/41	Ulcer had previously been excised and grafted without healing and amputation had been advised; ulcer healed rapidly after sympathectomy. Back at work with no pain	14 mo.
25	147393	M	27	Sickle-cell anemia; leg ulcer; syphilis	4 yr.	Right lumbar, 10/2/40	Ulcer had been twice excised and grafted but never stayed healed more than 3 months; healed after sympathectomy and stayed healed until traumatized 10 months later. Healed with grafts	1 yr.
26	224804	M	20	Sickle-cell anemia; leg ulcer; syphilis	9 yr.	Left lumbar, 3/6/41; skin grafts, right lumbar, 3/15/41	Ulcers recurred in 3 months	4 mo.

had chronic leg ulcers. In two instances in which the ulcers were very large and were associated with much induration, sympathectomy was combined with wide and deep excision of the ulcer and skin grafting. All of the ulcers healed promptly. In two there were slight recurrences which healed with pressure dressings. All of the patients who had had phlebitis had some degree of discomfort in the affected extremity, and in all of them the discomfort was relieved or diminished after operation. Cutaneous hyperesthesia was prominent in several, disappearing in all of them following operation. In some instances the edema was unchanged following operation, in some it has been less. Two patients were operated upon who had recurrent attacks of thrombophlebitis, in one of whom the attacks were of very long duration. One of these patients has continued to have attacks with almost as great frequency as before operation but they are now much milder and of shorter duration. The other patient has shown more improvement in this respect, having had only a single brief and mild attack during the twenty months since sympathectomy. This patient had an enormous elephantoid limb and was told before operation that reduction in size would almost certainly require a plastic operation of the type Homans has described. One accident occurred in this group. The patient in Case 17 had small pulmonary emboli after operation which were treated by ligation of the iliac veins. One patient had a very painful ulcer which developed in an uncomfortable edematous limb in which he had sustained a fracture of the fibula many years previously. There was no definite history of thrombophlebitis. His ulcer, which had been so indolent and painful that he had been tempted to accept his physician's advice and undergo amputation, healed promptly after sympathectomy and discomfort ceased.

Two patients with sickle-cell anemia and chronic leg ulcers were operated upon in an effort to learn whether sympathectomy might be of some aid in the solution of this very difficult problem. In neither could there be demonstrated any evidence of vasospasm before operation. One patient seems to have been helped somewhat; in the other the operation was apparently of no benefit.

Sympathectomy in Obliterative Arterial Disease.—Forty lumbar and three dorsal sympathectomies were performed upon twenty-eight patients with various sorts of obliterative arterial disease. Seventeen had definite or probable thromboangiitis obliterans; six had arteriosclerosis, four of them being diabetics; three had obliterative arterial disease following severe frostbite; one diabetic in whom there was no evidence of peripheral arteriosclerosis had an axillary arterial thrombosis; one patient had Werner's syndrome.

In Table III are grouped twenty-three sympathectomies on extremities in which there was a considerable element of vasospasm. Included are all those patients in whom occurred a rise of digital skin temperature to 28.5° C. or more after inhibition of vasoconstrictor impulses.

One case of Werner's syndrome is included. Because of the frequency of development of severe nutritional difficulties in the limbs of patients with this disease, one limb was sympathectomized in order to see whether with the passage of time this limb would fare better than the other. The answer to this question must wait for some years. Definite improvement was noted in all the others. The extremities have become warmer and of better color. Resting pain has been relieved in all instances save one. In this patient (Case 35) there was intense pain and numbness in a cold anesthetic foot which followed femoral arterial thrombosis. Sympathectomy was done five months after the vascular accident; although the circulation in the foot is significantly better than it was before operati .., the anesthesia, pain, and numbness persist. Whether regeneration will take place and relief of symptoms ensue one cannot say. Interestingly enough, another patient listed in Table IV had similar anesthesia and painful parethesias in a limb after axillary arterial thrombosis, underwent a sympathectomy six weeks after the occlusion, and within a month noted beginning return of sensation and has subsequently had complete relief of symptoms. Two patients complained of pain in the feet on exercise and both were relieved by sympathectomy. Five had intermittent claudication in the calves. One is unimproved, three are moderately improved, and one is apparently entirely relieved of this symptom. Five patients had ulcers or gangrenous digits. Healing occurred in three, and in two recently operated upon satisfactory healing seems to be taking place. Several who had had attacks of phlebitis have had no attacks since operation. No amputations have been necessary.

Case 29 is of particular interest. This 31-year-old patient with severe Buerger's disease had had an amputation of the left leg two years previously and for six months had had two ulcers of the lower portion of the right leg. Sympathectomy was followed by improvement in circulation and rapid healing of the ulcers. The patient continued to smoke to excess, to subject himself and especially his foot, to long exposure to cold, and in general to neglect his foot. For one year the circulation was excellent; then the foot began to get cooler and slightly cynanotic, and two small ulcers appeared. They are healing with bed rest. Another patient (Case 46, Table IV) is similarly interesting. He is a 47-year-old man with severe Buerger's disease who had migrating phlebitis, intermittent claudication, several ulcers of the left foot of a year's duration, and resting pain. He had undergone a lumbar sympathectomy fifteen months previously. The foot improved in color and warmth and the ulcers appeared to be healing when he left the hospital. He returned to work immediately and the ulcers failed to heal. One cannot say with certainty that these two patients would have done better had they had better care after operation, but I believe they illustrate the necessity for the best possible care after sympathectomy has been performed.

TABLE III

SYMPATHECTOMY IN CASES OF OBLITERATIVE ARTERIAL DISEASE WITH A CONSIDERABLE ELEMENT OF VASOSPASM

CASE NO.	UNIT NO.	SEX	AGE	DIAGNOSIS AND SYMPTOMS	DURATION OF SYMPTOMS	PROGNOSIS FROM SKIN TEMP. STUDIES	SYMPATHECTOMY, DATE	RESULT	PERIOD OF FOLLOW-UP
27	185417	M	29	Buerger's disease, coldness and numbness, resting pain, foot pain on exercise (1 block); unhealed stump from amputation left toe 4	2 yr.	Good	Left lumbar, 11/16/39 Right lumbar, 11/24/39	Ulcer healed, feet dry, warm, comfortable, walks well; working	6 mo.
28	209372	M	51	Buerger's disease and coldness, intermittent claudication (50 yds.); right femoral occlusion	4 yr.	Good	Right lumbar, 9/5/40 Left lumbar, 9/11/40	Feet warm; walking distance increased to 3½ blocks. No further improvement. Developed diabetes Dec., 1941	20 mo.
29	149430	M	31	Buerger's disease; left supracondylar amputation 2 yr. previously. Ulcers of right leg for 6 months	3 yr.	Fair	Right lumbar, 11/16/40	Ulcers healed rapidly; in spite of persistent smoking and neglect of foot, it remained well for 16 mo. then foot cooled and 2 small ulcers developed. Healing with rest	17 mo.
30	227575	M	33	Buerger's disease; gangrene of left foot, intermittent claudication (1 block)	6 mo.	Good right, nil left	Right lumbar, left Callander amputation, 4/18/41	Greatly improved; some aching leg and foot on first using artificial limb. Working	1 yr.
31	121897	M	38	Syphilis, tabes, and (?) Buerger's disease; feet cold and wet. Two perforating ulcers of right foot	5½ yr.	Excellent	Right lumbar, 4/25/41	Foot hot and dry; ulcer healed promptly and in spite of exceedingly poor foot care has remained healed	1 yr.
32	223153	M	55	Post-frostbite obliterative disease; feet cold, numb and painful. Has had pain in heels for years	5 mo.	Good	Left lumbar, 5/12/41 Right lumbar, 5/19/41	Symptoms disappeared except for discomfort in heels which is somewhat better	11 mo.

33	221757	M	43	Buerger's disease. Intermittent claudication (1 block)	6 mo.	Fair	Left lumbar, 5/5/41	Foot warm; walking distance increased to 3 blocks	11 mo.
34	226040	M	35	(?) Buerger's disease and pain and fatigue in calves unrelieved by elastic support; coldness and wetness of feet. Very small varices	4 yr.	Excellent	Left lumbar, 7/17/41 Right lumbar, 7/28/41	Feet warm and dry; discomfort persists but is considerably better	9 mo.
35	243185	M	59	Arteriosclerosis, left femoral arterial thrombosis, diabetes, intermittent claudication, resting pain and numbness; objective anesthesia foot and ankle	5 mo.	Good	Left lumbar, 10/30/41	Foot warmer and of better color, other symptoms unimproved; anesthesia unimproved	6 mo.
36	141314	M	50	Buerger's disease and syphilis; right foot cold and wet, claudication (1½ blocks). Recurrent thrombophlebitis of left leg.	10 yr. Claudication 1 mo.	Good	Right lumbar, 11/22/41 Left lumbar, 12/2/41	Feet warm, no claudication, no resting pain, no further attacks of phlebitis, no edema left leg	5 mo.
37	252612	M	43	Buerger's disease and migrating phlebitis; cold, wet feet. Resting pain.	5 yr.	Good	Left lumbar, 2/18/42 Right lumbar, 2/24/42	All symptoms relieved	2 mo.
38	252670	M	29	Buerger's disease, has lost parts of several digits from gangrene. Infected gangrene of the left first and third fingers	5 yr.	Fair	Left dorsal, 2/21/42 Right dorsal, 2/28/42	Hands warmer and comfortable; gangrenous digits well demarcated and free of infection	2 mo.
39	224159	M	67	Arteriosclerosis, gangrenous ulcer of right great toe with osteomyelitis; severe resting pain	5 wk.	Good	Right lumbar, 3/19/42	Pain relieved, distal end phalanx removed; healing	1 mo.
40	255252	M	52	Buerger's disease; pain and numbness walking or exposure to cold	4 yr.	Fair	Right lumbar, 3/24/42 Left lumbar, 3/28/42	Symptom-free; feet warm	1 mo.
41	177405	M	39	Werner's syndrome, obliterative arterial disease, scleroderma, calcinosis, and arteriosclerosis; ulcer over tendo achillis	?	Good	Right lumbar, 12/3/41	Foot warmer, otherwise no change; ulcer being treated by tubed graft	4 mo.

TABLE IV

SYMPATHECTOMY IN CASES OF OBLITERATIVE ARTERIAL DISEASE WITH LITTLE OR NO VASOSPASM

CASE NO.	UNIT NO.	SEX	AGE	DIAGNOSIS AND SYMPTOMS	DURATION OF SYMPTOMS	PROGNOSIS FROM SKIN TEMP. STUDIES	SYMPATHECTOMY, DATE	RESULT	PERIOD OF FOLLOW-UP
42	241492	M	46	Right axillary arterial thrombosis, mild diabetes, weakness, pain and numbness in hand and forearm, coldness, cyanosis, objective sensory loss	6 wk.	Poor to fair	Right dorsal, 10/6/41	Almost completely well. Warm hand, muscle power good, no pain, no objective sensory loss	6 mo.
43	224970	M	40	Buerger's disease; healed ulcer of right great toe, Coldness of right foot, pain in right foot on walking 3 blocks	8 mo.	Poor to fair	Left lumbar, 3/12/41 Right lumbar, 3/22/41	Feet warmer and of good color; very little discomfort, walks farther, still smoking	1 yr.
44	196805	F	62	Arteriosclerosis, diabetes, perforating ulcer on foot from puncture wound; no healing	6 mo.	Poor	Right lumbar, 12/22/41	Ulcer healed promptly and has remained healed	4 mo.
45	252670	M	35	Buerger's disease; lost left great toe from gangrene. Unhealed toes, stumps of right 2, 4, and 5. Extreme resting pain	4 yr.	Very poor	Right lumbar, 1/23/42 Left lumbar, 1/29/42	Pain completely relieved. Ulcers healed rapidly, feet warmer and of better color	3 mo.
46	220911	M	47	Buerger's disease; ulcers of left foot for 1 yr. Migrating phlebitis, resting pain, claudication (3-4 blocks)	12 yr.	(?) Unsatisfactory because of infection	Left lumbar, 1/29/41	Returned to work before ulcers healed; still unhealed, slight if any improvement	15 mo.
47	197311	M	34	Buerger's disease, severe resting pain, claudication, loss of right toes, 2, 3, 4, 5; unhealed stump	6 yr.	Nil	Right lumbar, 5/2/40 Left lumbar, 5/10/40	Pain relieved but no progress in healing of ulcer of right foot; right Callander amputation done 5/20/40. Left leg comfortable, foot slightly warmer and of better color	1 mo.

48	219569	M	20	Post-frostbite obliterative arterial disease, resting pain; gangrene of right great toe, cellulitis foot.	10 mo.	Nil	Right lumbar, 1/21/41	Toe amputated 2/18/41, tiny ulcer at site persists; occasionally some pain on walking, foot still cold. Patient feels he was helped considerably	15 mo.
49	222813	M	55	Post-frostbite obliterative arterial disease, loss of a number of toes from gangrene, ulcer of left foot, cellulitis and lymphangitis	22 yr.	Nil	Right lumbar, 3/21/41 Left lumbar, 3/31/41	Ulcer healed; feet slightly warmer and of better color	1 yr.
50	223436	M	25	Buerger's disease; very severe resting pain	9 mo.	Nil	Left lumbar, 3/13/41	Pain continued. Alcohol injection left posterior tibial nerve gave little relief. Patient persisted in smoking, refused further peripheral nerve section, and other treatment; left hospital to have amputation elsewhere	3 wk.
51	243448	M	63	Arteriosclerosis, diabetes, gangrene of distal end of first toe, with osteomyelitis; osteomyelitis of fifth toe	9 mo.	Nil	Left lumbar, 10/29/41 Amputation fifth toe, 12/3/41	Amputation stump healed per primam. Gangrenous ulcer of first toe healed	6 mo.
52	130061	M	58	Arteriosclerosis, claudication of right thigh (½ block); burning pain in foot and impending gangrene in toes	2 yr.	Nil left Good right	Bilateral lumbar, 11/19/41	Feet warmer and of better color; resting pain relieved but claudication unimproved	5 mo.
53	245441	M	57	Arteriosclerosis, claudication (1 block), resting pain, ulcer of right leg (4 months)	6 yr.	Nil	Right lumbar, 11/26/41 Left lumbar, 12/29/41	Pain relieved at first but returned after several weeks as ulcer increased in size; right Callander operation done 12/29/41. Patient now comfortable.	4 mo.
54	255653	M	43	Buerger's disease; unhealed amputation stump of right fourth toe. Impending gangrene of right toes 2 and 3. Severe resting pain	4 mo.	Nil	Right lumbar, 3/23/42 Left lumbar, 3/28/42	Marked pain relief, ulcer healed; increased warmth and better color	1 mo.

In Table IV are listed sympathectomies on twenty extremities in which preoperative studies demonstrated that the circulatory deficiency was due entirely or almost entirely to occlusive disease with little or no element of vasospasm. These patients are the sort for whom sympathectomy has ordinarily been felt to offer no benefit. No definite promise of help was offered to any of them, and the operation was carried out with the understanding that it was done more or less as an experimental procedure. The results are very interesting. Case 46 has already been discussed. There were several other poor results. Case 48 is that of a young man who had had a severe frostbite and who had pronounced obliterative disease with gangrene of one toe, cellulitis of the foot, and severe pain. After sympathectomy the toe was amputated. There remains a tiny ulcer at the site of amputation and the foot is still cold, but the patient has pain on walking only occasionally and feels that he was helped a great deal.

Case 50 in this group is that of a young man with rapidly progressive Buerger's disease and severe resting pain who was not helped by sympathectomy. There was some relief from alcohol injection of the posterior tibial nerve, but the patient refused other peripheral nerve interruption or other treatment, persisted in smoking heavily, and left the hospital determined to have an amputation of the leg. One patient (Case 53) with arteriosclerosis, intermittent claudication, an infected ulcer of the leg, and severe resting pain had complete relief of pain for several weeks following sympathectomy. The pain returned, however, after his discharge, the ulcer failed to heal, and amputation was carried out. The other leg has been entirely comfortable since operation. One patient (Case 47) was an unfortunate young man with Buerger's disease, intermittent claudication, and severe resting pain who had undergone amputation of four toes with failure of healing of two, hundreds of hours of pavex therapy, intermittent venous occlusion, the intravenous administration of hypertonic salt solution, alcohol injection of peripheral nerves, and other measures during the past six years, during which time his disability progressed steadily. There was no demonstrable element of vasospasm, and right lumbar sympathectomy was performed at his request as an experimental procedure. There was almost complete relief of pain but no progress in healing of the ulcers. Because of the relief of pain the other extremity was sympathectomized, with good result. Amputation of the right leg was necessary.

In the other cases the result was surprisingly good. The complete relief of symptoms in Case 42 has been discussed. Cases 45 and 54, which appeared hopeless, had relief of very severe pain, healing of ulcers, and improvement in warmth and color. In Case 44, a diabetic with poor circulation and a perforating ulcer of the sole of the foot which had showed no improvement during six months of good treatment, healing took place promptly after operation. In another patient (Case 51) with diabetes and severe arteriosclerosis there was healing of a

gangrenous toe in which there was x-ray evidence of osteomyelitis and primary healing of the amputation stump following removal of another toe in which there was extensive chronic osteomyelitis. Case 49 is that of a man who, during a period of 22 years, had gradually lost parts of most of his toes from gangrene following a severe frostbite. A chronic ulcer of the foot healed promptly after operation. One patient (Case 52) with arteriosclerosis, intermittent claudication in the thigh, burning pain in the foot, and impending gangrene of several toes has had noticeable improvement in circulation and relief from resting pain. The claudication continues. Case 43 is a man with Buerger's disease who complained of coldness of his foot and pain on walking a few blocks. He showed definite improvement in circulation following operation and could walk without discomfort.

It should be stated that almost all of the patients with obliterative arterial disease had had the usual nonoperative treatment before operation—exercises, pavex, intermittent venous occlusion, intravenous administration of saline solution, intravenous sodium thiosulfate injections, and abstinence from smoking. Some had undergone prolonged and intensive treatment. It is also of interest that various forms of nonoperative treatment have been tried since operation in a few of the patients who had residual symptoms without any further improvement.

DISCUSSION

Sympathectomy should receive full consideration as a means for the treatment of peripheral vascular disease. Every patient with obliterative arterial disease of the extremities and those with annoying vasospastic disease merit careful study to see whether they may be helped by sympathectomy. Before any patient is subjected to sympathectomy he should be studied carefully in an effort to evaluate the possible benefit from this procedure from the standpoint of release of vasospasm, relief of pain, and reduction in heat loss through sweating and evaporation. Sympathectomy will probably find a wider field of applicability in the treatment of other disorders of the peripheral circulation.

No matter how striking the improvement after sympathectomy, patients should continue to take the best of care of their extremities and to do everything possible to prevent progress of the disease and to further the development of collateral circulation. Those with arterial circulatory deficiency should not smoke, should avoid exposure to extremes of temperature, should take exercise to the point of tolerance, and should take special exercises to promote collateral circulation. Every patient who has undergone sympathectomy should keep the extremity well greased in order to avoid excessive dryness of the skin.

Sympathectomy gives excellent results in the treatment of Raynaud's disease and the other purely vasospastic arterial circulatory deficiencies. Ordinarily, the extremities become warm and dry and attacks of vasospasm cease. In a small percentage of cases, as in one

of mine, there are no further attacks from emotional stimuli but the extremities continue to cool abnormally on exposure to cold. The proper explanation for this small group of less favorable cases is difficult to find. The current tendency is to ascribe the result to too much "local fault." In the vasospastic disorders, as in most diseases, the earlier the stage at which treatment is undertaken the better the result obtained. Results are certainly better in those cases in which there are no pathologic alterations in skin or bone. In the present series all instances of scleroderma were improved, in some patients very strikingly. There was no further spread, the skin became softer, contracted stiff fingers became more movable. There are cases, however, in which less favorable results are obtained. It seems fair to say that improvement will take place in scleroderma in most instances, that in some there will be little improvement but the progress of the disease will be stopped, and that in others the progress of the disease will be definitely slowed. Digital ulcers may be expected to heal and not to recur.

In the common obliterative arterial diseases, thromboangiitis and arteriosclerosis, much can be accomplished by sympathectomy. The circulation often improves noticeably, sweating ceases and with it the tendency to maceration and to epidermophytosis, ulcers often heal, and impending gangrene frequently disappears. There is commonly the most gratifying relief of resting pain. If the relief should not be complete, judicious application of peripheral nerve interruption, as suggested by White, will prove of great benefit. Pain on exercise presents a more difficult problem. The pain which some patients experience in their feet on walking is ordinarily relieved. The typical intermittent claudication of the calves, however, is the symptom for which sympathectomy accomplishes least. Sometimes there is no improvement at all in the distance the patient can walk, although actual pain may be replaced by a sense of fatigue. Occasionally there is striking relief of intermittent claudication. More often there is moderate increase in the distance walked before onset of pain. To increase the walking distance from half a block to three or four blocks is not a spectacular improvement, but it may mean the difference between a very restricted life and a fairly active one.

In general, the good effects which may be expected from sympathectomy can be fairly well assayed by careful study before operation. Whenever there is disagreement between the evaluation arrived at from preoperative studies and the actual result of operation, the result obtained is always better than the one anticipated. Experiences such as those recorded in Table IV illustrate the fact that certain patients in whom no significant element of vasospasm can be demonstrated may profit remarkably by sympathectomy. Consequently, I feel that in cases of apparently pure or almost pure obliterative arterial deficiency certain ones which appear hopeful to those experienced in this field may justifiably be subjected to sympathectomy. It must be remembered that this is a relatively safe operation with a short and comfortable con-

valescence. If a number of major amputations can be saved in this manner one need not regret a few futile efforts. It should be stated emphatically, however, that the routine use of sympathectomy without any attempt at selection of cases and without proper preoperative study would be most inadvisable and would only bring discredit to the procedure. It is my impression that in any patient with obliterative arterial disease and significant vasospasm, one can accomplish fully as much, probably more, with a short period of hospitalization and a simple operative procedure as one can hope to accomplish by almost daily treatments with the pavex boot, intermittent venous occlusion, various sorts of intravenous injections, and other so-called conservative measures. The timesaving and moneysaving element of this choice of treatment is a most important consideration to most of the patients, many of whom come for help only after they have become impoverished by prolonged medical treatment and inability to work.

Patients with sequelae from frostbite are an interesting group. Some of them have no demonstrable evidence of obliterative disease but present a more or less typical Raynaud syndrome. Such patients respond beautifully to sympathectomy. Others have varying degrees of obliterative arterial disease. The role of sympathectomy in their treatment is much the same as in the other obliterative arterial diseases. Proper treatment at the time of the initial frostbite will undoubtedly reduce greatly the number of patients who have vascular symptoms thereafter. It is my feeling that lumbar sympathetic procaine block is probably the most effectual treatment for such cases. If one or more such injections should fail to give lasting benefit one should not hesitate to proceed directly with operative sympathectomy.

Similarly, the proper care of those suffering from acute thrombophlebitis will undoubtedly reduce the number who develop phlebitic sequelae. Sympathetic block, suggested by Leriche and introduced in this country by Ochsner and his associates, is a valuable aid in the treatment of acute thrombophlebitis. Proper and sufficient bed rest until there is little or no edema when the affected limb is dependent, and the use of elastic support as long as there is the slightest edema are other important measures. Such care will decrease the number of patients who will subsequently suffer from swollen painful limbs, intractable ulcers, hyperesthesia, and recurrent episodes of thrombophlebitis. Sympathectomy seems to have a definite place in the treatment of certain of these sequelae, although its status is in certain respects still in the experimental stage. From my experience with two cases it is not felt that sympathectomy will prevent further attacks in cases of recurrent thrombophlebitis, but it can be expected to decrease the duration and severity of attacks. Perhaps it may prove useful in this regard in combination with ligation of the vein. Sympathectomy affords considerable or complete relief from the annoying discomfort that so commonly exists after phlebitis, especially in those patients with cutaneous hyperesthesia and vasospasm. It has a definite place in the man-

agement of postphlebitic ulcers, especially when they are small and the surrounding tissue is not too indurated. In cases of large ulcers with much induration, wide deep excision of the affected tissue and skin grafting should be carried out. If such patients have demonstrable vasospasm this procedure may be combined profitably with sympathetic denervation.

Sympathectomy gives excellent results in certain cases of causalgia. In cases of post-traumatic edema and osteoporosis and other vasospastic states, as well as in "phantom-limb pain," it should be carried out if relief has been obtained with procaine block of the sympathetic nerves provided a permanent cure cannot be effected by one or several procaine injections.

SUMMARY

The value of sympathectomy in the treatment of disorders of the peripheral circulation has been discussed. The study of patients in an attempt to evaluate the possible benefit of sympathectomy is outlined, the operative technique of dorsal and lumbar sympathectomy is described, and the results in sympathetic denervation of eighty-three extremities are presented.

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MEASUREMENT OF THE CIRCULATION RATE

A Review of the Methods

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INTRODUCTION

THE methods proposed for determining the rate of blood flow in the vessels are numerous, and one confronted with the task of measuring this velocity must evaluate carefully all the factors involved in the particular type of cases to be studied. Circulation rate, which is the time required for blood to be transported from one point in a vessel to a definite distant point, is expressed most accurately in terms of distance per unit time, as centimeters per second. Clinically, however, the circulation rate has come to mean just the time required for the transport, in order to differentiate it from the circulation time, which has been defined by Koch:[47] "By circulation time is understood the time required for a blood particle to traverse an entire circulation." Obviously, since the circulation time may be long or short depending on the choice of the vessels selected by the indicator, circulation time is rarely determined clinically.

Blumgart[5] has written a good discussion of blood velocity, and with his collaborators[69] has stated that, in order to be used in testing the circulation rate, the indicator should: (a) be nontoxic in the amounts used, (b) not itself influence the velocity of flow at the time of determination, (c) with its effect, disappear rapidly, (d) have a short reaction time, and (e) be applicable to normal and pathologic cases.

The following review is intended as a survey of the history of circulation rate determination.

METHODS

Volkmann, in 1850, used the pressure of the blood as a measure of its velocity, replacing a segment of the blood vessel with a hairpin-shaped glass tube filled with water, an instrument to which he applied the name haemodromometer. Vierordt, in 1858, inserted a pendulum-like device in the vessel lumen, the velocity being calculated from the extent of deflection, an instrument he called the haemotachometer, and which was later modified by Chauveau, Bertolus, and Laroyen.[12] Cybulski's photohemotachometer[14] was a variation of the same principle. Similarly, the differential manometers of Frank[22] and Daly,[15] though differing from each other in principle, have the common disadvantage of requiring insertion into a divided vessel. The stromuhr, even in the experimental

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animal, was proved by Dogiel[16] to be inaccurate, the velocity varying from turn to turn. By prolonging the time of observation, however, Burton-Opitz[11] was able to use the recording stromuhr successfully. The thermostromuhr of Rein[57] stands as a magnificent tribute to the genius of its inventor, blood volume flow between two points in a vessel being calculated from the thermal change after a definite quantity of heat has been applied to the vessel wall.

These devices, however suitable for the experimental animal, cannot be employed clinically, and investigators have suggested numerous methods by means of which circulation rate may be determined with less trauma, and, perhaps, with more directness. Vierordt[67] previously had made use of the "muscae volitantes" of Aristotle, measuring the capillary blood velocity in his own eye by projection of the image of a moving corpuscle on a white surface and obtaining a rate of 0.5 mm. per second as capillary blood velocity. Even before this, Hering[33] had determined the jugular-to-jugular time in a horse by injecting potassium ferrocyanid into one jugular vein and determining the time of arrival in the opposite jugular by the Prussian blue reaction, finding that the jugular-to-jugular and jugular-to-tarsal times were nearly the same. Vierordt, however, obtained the blood samples for testing in little cups fixed to a disk rotating at a definite rate, an instrument he described as the hemotachometer, a method which was highly refined in the hands of Hermann[34, 35] and Ainser and Lohe.[1] Very accurate determinations of circulation rate between different arbitrary points in the experimental animal have been made by these investigators.

Using this principle, many substances have been utilized. Koch[46] injected fluorescein into one vessel and collected blood samples on a Vierordt disk. Meyer[53] used methemoglobin obtained from a donor animal, determining its arrival time spectroscopically, and Thompson, Alper, and Thompson[65] injected brilliant vital red solution in man, securing blood samples from the same vein every fifteen seconds. Similarly, Klein and Heinemann[45] used Congo red solution, Heilmeyer and Riemschneider[31] determining the instant of arrival spectroscopically. Stewart[64] injected methylene blue into the rabbit vein and determined its arrival time in the retina by direct inspection. Bornstein[10] administered a single deep inspiration of air containing 5 to 7 per cent carbon dioxide, and determined the interval between the inspiration and the first deep inspiration to follow involuntarily, assuming this interval to be circulation time between the lung capillaries and the respiratory center. Franck and Alwens[21] roentgenographically determined the arrival of bismuth oil at a second point in a vessel after injection at a first point. Frimann-Dahl[23] measured the interval between the injection of Per-Abrodil, a radiopaque iodine-containing substance, into the saphenous vein and its roentgenographic appearance in the femoral vein. Hirschsohn and Maendel[43] and Kahler[44] used the intravenous injection of 5 c.c. of a 10 per cent solution of calcium chloride, which was followed

by a feeling of warmth and burning in the throat, and which they believed was due to the arrival of the salt at the nerve endings in the tissues, the warm feeling gradually extending to the chest, abdomen, pubis, arms, legs, and feet, in an inconstant order. Leschke[48] used calcium bromide for this purpose with a similar effect.

Blumgart and his co-workers[6, 9] studied circulation rate by injecting radium C at one point and determining its arrival at another point by a counting chamber, an exceedingly accurate clinical method but one equally tedious and elaborate. Edmunds[17] and Stewart,[61-63] however, had injected concentrated sodium chloride solution intravenously into animals and determined its arrival at one point by the change in conductivity of the vessel placed between two nonpolarizable electrodes. Meldolesi applied this method to human beings in 1925, using an electrode placed near the observation point in a galvanometer circuit.

Neubauer,[54] experimenting on the choleretic action of bile salts, found that a side action of dehydrocholic acid was its bitter taste, and Winternitz, Deutsch, and Brüll[71] using this substance as "decholin" first described its action as a determiner of circulation time to the tongue, a method which Gargill[24] has tested and found to be practicable. Eppinger[18] first recognized the applicability to this problem of the skin flushing after intravenous histamine injection, the method having been used clinically in over 600 determinations by Wollheim and Lange.[72] The principal objection to this method, observed by Harmer and Harris,[29] aside from its limited observable circuit, is the fall in blood pressure and great increase in pulse rate which accompanies the reaction. Fishberg, Hitzig, and King[20] found that saccharine introduced into a vein caused a sweet taste, the interval between injection and the signal by the patient being the time for passage through the vein, to and through the heart and lungs to the tongue. The reaction time of the patient is included. Webb, Sheinfeld, and Colin[68] measured the interval between the intravenous injection of a small quantity of ether and its detection in the breath of the subject by the observer.

Geppert, in 1889,[26] observed an increase in respiratory depth in animals after intravenous injection of calcium cyanide during his studies on the mechanism of hydrocyanic acid asphyxiation. Gasser and Loevenhart[25] then used sodium cyanide as a respiratory stimulant in animals in an attempt to show that stimulation of the respiratory center was due to diminished oxidation, observing that the respiratory response in the rabbit after external jugular injection was "surprisingly rapid" (within three seconds). A few years later, Loevenhart and his colleagues[52] applied the method clinically, Robb and Weiss[58] finding later that the toxic dose of sodium cyanide was twenty times that required for a respiratory response. These workers used a 2 per cent solution in water and concluded that the cyanide was excreted as hydrogen cyanide by the lungs, as cyanides and sulfocyanides by the urine, and that the remainder was converted into relatively innocuous sulfur compounds.

The question as to the exact site of action of sodium cyanide in producing the hyperpnea has been debated. The fact that HCN is one of the strongest respiratory stimulants known (Heinz[32]) has led some to believe that this action of cyanide is directly on the respiratory center (Robertson;[59] Loevenhart and associates;[28, 49, 51] Gesell and co-workers;[27, 56, 70] and Heffter[30]). However, in 1927, Hertzman and Gesell[36] in cross circulation experiments had evidence that cyanide could exert its stimulating action with the head isolated. This work was followed

TABLE I

CIRCULATION TIMES AS DETERMINED BY VARIOUS OBSERVERS WITH DIFFERENT METHODS (TIMES IN SECONDS)

TIME	ROUTE	METHOD	OBSERVER
20-25	jugular-to-jugular (horse)	ferrocyanide	Hering[33]
14-59	jugular-to-jugular	ferrocyanide	Ainser and Lohe[1]
18-23	cubital-to-cubital (man)	fluorescein	Koch[46, 47]
16-18	femoral-to-right atrium (dog)	bismuth oil	Franck and Alwens[21]
9-15	cubital-to-mouth (man)	calcium chloride	Kahler[44]
10-12	cubital-to-skin (man)	calcium chloride and bromide	Leschke[48]
6	cubital-to-opposite radial artery (man)	Congo red	Klein and Heinemann[45]
4-7.6	lesser circulation (dog)	conductivity	Romm[60]
3.5-7.1	greater circulation (dog)	conductivity	Romm[60]
8.9-12.8	entire circulation (dog)	conductivity	Romm[60]
13-30	cubital-to-face	histamine	Weiss, Robb, and Blumgart[69]
14-24	cubital-to-opposite brachial (man)	radium C	Weiss, Robb, and Blumgart[69]
15-20	cubital-to-tongue (man)	decholin	Gargill[24]
8-14	cubital-to-tongue (man)	decholin	Winternitz et al.[71]
12.5-18	cubital-to-carotid (man)	sodium cyanide	Gargill[24]
8-16	femoral vein-to-carotid (dog)	sodium cyanide	Winder, Winder and Gesell[70]
1	through heart (man)	radium C	Blumgart and Weiss[7]
6.5	lesser circulation (man)	radium C	Blumgart and Weiss[8]
10.7	cubital-to-tongue (man)	saccharine	Webb, Sheinfeld, and Colin[68]
6.3	cubital-to-lung (man)	ether	Webb, Sheinfeld, and Colin[68]

by the painstaking, ingenious studies of Heymans and his collaborators,[37-42] who have proved that the carotid sinus is the origin of the respiratory reflexes, that in the dog cyanide acts on the sinus with little or no effect on the center, that there is an instantaneous effect on respiration after injection of cyanide into the common carotid artery, that the action of cyanide on the center is depressive, and that no response to

cyanide is obtained if both carotid sinuses are denervated. These researches have been confirmed by Bernthal[4] and Comroe and Schmidt.[13]

The only objections to the use of sodium cyanide in the determination of circulation rate relate to the effect on the heart, which may alter the true rate by the salt itself. Barnard,[2] Loevenhart and Eyster,[50] Evans,[19] and Bellis[3] have shown that cyanide increases the force, rate, and amplitude of the heart, but these effects occur simultaneously with the respiratory response and cannot, therefore, be taken to influence it. Another objection to the methods of determining circulation rate by intravenous indicators such as sodium cyanide arises from the observations of Tigerstedt[66] that the speed of the central or axial portions of a stream may be twice that of the peripheral portions, but this, although true for constant flow in rigid tubes of constant diameter, does not hold for the blood stream with its turbulence due to pulse, elastic walls, contained cells, and varying diameters of the vessels.

By way of comparison, Table I is presented to show the values obtained for circulation rates by the corresponding observers using various methods. The purely objective response obtained after the injection of sodium cyanide intravenously greatly simplifies its routine use. With this advantage is coupled that of application in colored individuals (where histamine cannot be used).

SUMMARY

A brief historical review of the methods which have been used for the determination of the circulation rate has been presented. This information formed the basis for selecting sodium cyanide as the determinant in surgical patients. These results are described in a succeeding paper.

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THE CIRCULATION RATE AFTER OPERATION

With Special Reference to the Effect of Position

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INTRODUCTION

IT MUST be evident, to even a casual observer, that opening the abdomen upsets a finely adjusted circulation-respiration equilibrium, the delicate interplay of whose components is responsible for normal circulatory efficiency. As a group, therefore, surgeons are, perhaps, more interested in acute changes in circulatory dynamics, and with good reason, for circulatory collapse, during surgery or immediately after, points an accusing finger at the surgeon. It is well known that the human body has an almost incredible, but limited, power to adjust itself to unusual demands on its circulation. In fact, Harvey[10] wrote, "The circuit of blood is accomplished now more rapidly, now more slowly according to the temperament, age, and so forth of the individual, to external and internal circumstances, to naturals and non-naturals—sleep, rest, food, exercise, affections of the mind, and the like—."

Frimann-Dahl,[9] who has advocated the theory of slowed venous flow as the most important factor in postoperative thrombosis, showed roentgenographically that the movements of the diaphragm, which acts as a pump on the veins inferior to it, are shallower after operation; especially is this decreased pumping action evident on the veins of the upper abdomen. Men were more affected in this manner than were women, but the effect in women was more protracted. He found that the saphenous-to-femoral venous flow in patients undergoing operations predisjosing to thrombosis was reduced to a standstill. Flow in the arm veins, however, was not so retarded. Postoperative distention caused considerable venous stasis. He, therefore, attributed the slowing of saphenous flow to (a) abdominal distention leading to increased intra-abdominal pressure and to (b) inactivity of patients. Bellis and Wangensteen[1] have confirmed the retarding effect of intestinal distention on venous return from the lower extremities in man and in the experimental animal.

Lanzara[16] determined the decholin time in sixty-six cases preoperatively and postoperatively. He usually found a shortened postoperative time, the figures remaining within normal limits, however.

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Webb, Sheinfeld, and Colin[30] stated that no significant differences between preoperative and postoperative circulation rates were present when determined by the saccharin and ether method. They also stated that laparotomy, spinal anesthesia, morphine, slight elevation of pulse and temperature, and moderate abdominal distention did not affect the circulation rate. Inspection of their table of eight cases subjected to spinal anesthesia, however, shows that every patient experienced a shortened circulation time after the procedure. In one individual, the time after the anesthetic was nine seconds shorter than before.

Since the effect of operation on circulation rate is apparently open to discussion, the question was reopened in the investigations to be described.

METHOD

Using the sodium cyanide method, the cubital-to-carotid and ankle-to-carotid circulation times were determined in 191 cases preoperatively

TABLE IA

CUBITAL VEIN TO CAROTID BODY AND ANKLE VEIN TO CAROTID BODY CIRCULATION TIMES (SECONDS) DETERMINED PREOPERATIVELY AND POSTOPERATIVELY BY THE SODIUM CYANIDE METHOD

TYPE OF OPERATION	NO. OF CASES	CUBITAL-TO-CAROTID*		ANKLE-TO-CAROTID*	
		PRE-OPERATIVE	POST-OPERATIVE	PRE-OPERATIVE	POST-OPERATIVE
Appendectomy	21	13 (8-20)	10 (8-15)	30 (20-41)	28 (18-47)
Cholecystectomy	18	15 (8-26)	12 (8-24)	35 (20-63)	28 (18-45)
Neck dissection and thyroidectomy	17	15 (9-28)	14 (9-29)	39 (25-67)	35 (27-41)
Craniotomy	15	20 (11-35)	15 (13-20)	36 (28-42)	34 (22-45)
Herniorrhaphy	14	15 (8-25)	13 (7-23)	31 (17-58)	38 (18-65)
Colostomy	8	25 (8-92)	15 (10-25)	38 (28-54)	34 (23-60)
Kidney-ureter	7	17 (11-32)	16 (12-21)	30 (15-45)	28 (24-31)
Exploratory laparotomy	7	17 (11-30)	15 (10-14)	38 (30-46)	31 (27-35)
Hemorrhoidectomy	7	13 (8-19)	12 (8-17)	30 (25-45)	33 (23-47)
Gastric resection	6	23 (13-30)	16 (10-17)	48 (30-85)	30 (23-42)
Posterior rectectomy	6	15 (10-17)	14 (8-19)	37 (26-52)	30 (20-37)
Radical mastectomy	6	12 (8-16)	12 (8-17)	31 (20-49)	30 (24-36)
Miscellaneous†	59	16 (7-31)	14 (8-31)	41 (15-95)	38 (21-80)
Total	191	17 (7-92)	14 (7-31)	36 (15-95)	32 (18-80)

*Range enclosed in parenthesis below average for each group.

†Lumbar sympathectomy, transurethral resection, breast biopsy, suprapubic cystostomy, rectal polypectomy, drainage ischiorectal abscess, cardiolysis, perineal prostatectomy, gastroenterostomy presacral neurectomy, drainage pancreatic abscess, lip excision, splenectomy, amputation of penis, enterostomy, leg amputation, closure perforated ulcer, excision mandible, axillary dissection, scrotal-crural fixation, ligation external carotid, etc.

TABLE IB

CASE	PREOPERATIVE				POSTOPERATIVE			
	CUBITAL-TO-CAROTID	ANKLE-TO-CAROTID	PULSE	TEMPERATURE	CUBITAL-TO-CAROTID	ANKLE-TO-CAROTID	PULSE	TEMPERATURE
1. Appendectomy								
1	13	--	82	99.0	12	--	82	96.6
2	11	--	100	98.0	8	--	106	100.0
3	13	29	68	98.6	10	29	104	104.4
4	12	22	100	99.2	15	23	96	99.4
5	9	28	104	98.6	9	30	86	100.0
6	12	--	72	97.2	11	--	80	99.2
7	12	20	92	--	9	20	128	98.8
8	20	36	60	--	11	40	--	--
9	--	30	100	99.0	--	47	80	98.8
10	11	25	80	98.0	11	18	80	100.0
11	20	41	96	98.0	12	30	82	99.4
12	12	37	--	--	12	35	80	--
13	10	22	68	97.8	9	19	100	99.8
14	8	20	88	99.3	7	22	100	100.6
15	15	38	76	--	11	33	83	--
16	16	26	80	98.6	8	20	102	99.0
17	10	41	72	98.4	10	34	84	99.8
18	13	33	74	99.0	10	30	76	100.2
19	12	28	68	98.6	13	20	80	100.0
20	9	38	82	--	10	--	94	--
21	12	27	80	--	8	--	80	--
Avg.	13	30			10	28		
2. Cholecystectomy, Choledochotomy, and Cholecyst-gastrostomy								
1	10	35	84	99.2	10	18	92	100.2
2	18	--	88	98.6	14	--	132	101.8
3	26	63	72	98.8	24	45	64	100.0
4	12	25	80	98.0	10	30	96	98.6
5	10	28	84	98.6	12	28	88	99.0
6	13	25	84	98.2	16	23	92	98.8
7	12	20	80	97.8	8	24	112	99.8
8	18	40	68	98.6	12	20	100	101.0
9	15	--	60	98.6	10	--	--	--
10	10	33	112	98.4	10	22	96	100.4
11	23	32	100	98.4	12	24	104	100.0
12	23	--	70	97.4	24	--	--	--
13	14	36	78	99.4	10	33	76	100.0
14	14	40	74	98.8	12	25	78	99.8
15	17	35	74	97.8	9	31	88	99.4
16	11	43	76	98.0	10	38	78	100.2
17	8	--	82	97.8	9	--	100	--
18	15	--	--	--	11	--	--	--
Avg.	15	35			12	28		
3. Thyroidectomy and Neck Dissection								
1	28	67	72	97.8	23	40	60	98.4
2	11	--	104	99.0	11	--	--	--
3	28	--	78	98.4	29	--	--	--
4	17	26	84	98.0	15	27	--	--
5	9	25	84	99.6	14	31	92	99.0
6	10	27	104	97.4	12	27	84	98.2
7	15	--	--	--	15	--	--	--
8	12	--	--	--	16	--	--	--
9	14	36	--	--	14	42	--	--
10	17	--	--	--	16	--	--	--
11	12	--	80	98.8	10	--	100	99.8
12	10	38	76	99.0	10	39	88	100.2
13	14	45	78	99.4	9	40	104	100.6
14	13	40	90	--	12	--	110	--
15	13	--	74	--	11	28	106	--
16	14	45	76	98.6	12	41	85	99.8
17	10	40	70	97.8	10	33	78	100.0
Avg.	15	39			14	35		

TABLE 1B—CONT'D

CASE	PREOPERATIVE				POSTOPERATIVE			
	CUBITAL-TO-CAROTID	ANKLE-TO-CAROTID	PULSE	TEMPERATURE	CUBITAL-TO-CAROTID	ANKLE-TO-CAROTID	PULSE	TEMPERATURE
4. Craniotomy (including ventriculograms, explorations, and trigeminal rhizotomies)								
1	35	--	86	99.0	20	--	104	--
2	21	42	86	98.6	15	45	72	--
3	21	28	88	97.4	15	32	84	99.8
4	24	—	80	97.4	13	—	80	98.8
5	15	28	92	98.0	14	23	94	100.8
6	28	35	72	98.6	20	31	--	--
7	20	--	--	--	15	--	80	99.6
8	11	29	80	98.6	14	22	100	101.2
9	29	42	68	97.8	14	42	80	100.6
10	14	--	64	98.0	15	--	76	98.6
11	22	--	64	98.0	17	--	92	101.0
12	16	--	136	99.2	13	--	100	100.8
13	14	39	82	--	14	39	76	--
14	16	38	78	99.0	18	40	80	100.4
15	11	--	--	--	10	--	--	--
Avg.	20	36			15	34		
5. Herniorrhaphy (inguinal and ventral)								
1	18	42	78	98.6	14	40	76	--
2	15	--	88	99.0	12	--	118	100.0
3	12	18	100	--	12	16	--	--
4	9	--	72	98.0	7	--	82	99.6
5	17	28	68	98.6	10	53	88	100.6
6	25	55	74	98.4	23	54	60	98.6
7	8	17	132	98.8	10	18	131	100.4
8	14	33	76	98.0	13	39	80	99.2
9	14	35	68	98.6	10	35	88	98.6
10	22	31	52	97.0	19	65	76	100.0
11	13	--	76	98.6	15	--	--	--
12	18	--	76	99.2	16	--	--	--
13	13	22	84	98.0	11	23	86	100.4
14	13	--	--	--	10	--	--	--
Avg.	15	31			13	38		
6. Colostomy								
1	92	39	68	98.6	25	33	80	99.4
2	17	40	80	98.4	17	37	74	99.0
3	18	27	92	--	15	25	--	--
4	17	47	84	97.4	15	37	--	--
5	15	32	72	100.2	13	23	--	--
6	24	54	76	96.4	12	60	76	99.4
7	11	28	--	--	10	24	--	--
8	8	--	--	--	10	--	--	--
Avg.	25	38			15	34		
7. Kidney and Ureter (including pelviolithotomy, ureterolithotomy, nephrostomy, and ureteropelvioplasty)								
1	32	45	80	98.4	20	30	80	100.2
2	18	37	80	98.6	21	27	120	98.6
3	15	30	84	99.0	15	24	120	102.0
4	11	16	100	98.0	17	24	--	--
5	16	15	100	98.2	12	31	112	98.2
6	12	35	80	--	12	28	86	--
7	12	34	80	99.4	12	35	84	100.8
Avg.	17	30			16	28		
8. Exploratory Laparotomy								
1	15	30	96	97.2	13	28	--	--
2	13	36	72	97.4	13	34	--	--
3	17	40	72	--	12	27	108	98.8
4	15	35	96	98.2	8	23	124	99.8
5	30	40	76	98.6	19	30	72	99.0
6	15	46	--	--	10	44	--	--
7	12	37	--	--	10	35	--	--
Avg.	17	38			15	31		

TABLE IB—CONT'D

CASE	PREOPERATIVE				POSTOPERATIVE			
	CUBITAL-TO-CAROTID	ANKLE-TO-CAROTID	PULSE	TEMPERATURE	CUBITAL-TO-CAROTID	ANKLE-TO-CAROTID	PULSE	TEMPERATURE
9. Hemorrhoidectomy								
1	13	45	78	98.2	8	47	80	99.4
2	9	27	82	98.6	11	27	88	100.2
3	9	30	--	--	--	44	--	--
4	8	25	--	--	16	23	--	--
5	14	28	80	100.0	17	26	82	99.8
6	16	30	76	99.0	9	35	86	101.0
7	19	26	74	98.8	11	31	100	100.2
Avg.	13	30			12	33		

and postoperatively. The first determination in each individual was made about twenty hours before operation, the second determination about twenty hours after operation. Of the cases listed (Table I, *A*), 132 could be classified in groups according to operative procedure; 59 were miscellaneous procedures. The type of anesthetic employed varied, and many of the operations were not laparotomies. "Nervous" patients, those with varices of the lower extremities, and children were not investigated. Every determination was made with the trunk horizontal and the extremities in the plane of the trunk, the veins into which the injections were made being approximately at heart level, and the patient instructed against muscular movement. It was frequently necessary to elevate the injected extremity by a pillow in order to bring the vein used to the level of the anterior axillary fold.

Because the organism seems to respond better to a second stimulus by sodium cyanide than to a first, the response to injection was first determined from one of the cubital veins, and following a five-minute interval (the time required for the destruction and elimination of the salt), an injection into one of the ankle veins was made. The cubital-to-carotid time or ankle-to-carotid time is the time recorded by an observer with a stop watch between the intravenous injection and the first deep inspiration to follow. This includes the time required for the stimulant to travel from the intravenous needle point to and through the heart and pulmonary circulation to the carotid body. Although Loevenhart, Lorenz, Martin, and Malone[18] used injections of 3 to 5 mg. of sodium cyanide, we used $\frac{2}{10}$ to $\frac{5}{10}$ of a c.c. of a 2 per cent solution (4 to 10 mg.) for the arm vein and $\frac{5}{10}$ to 1 c.c. (10 to 20 mg.) for the ankle vein, the amount varying principally with the patient's apparent weight. In no case was the patient informed of the response expected, this rule being adhered to in order to avoid any subjective influence. Occasionally, the response is associated with symptoms suggestive of motor irritation, lasting less than a minute; in some cases a slight temporary nausea was noted, but in none was there any alarming sequela. Care was taken to allow none of the solution to be injected extravascularly, since such an accident may be followed by a local necrosis.

EFFECT OF POSITION ON CIRCULATION RATE

In order to study the effect of position of an extremity on the circulation rate of sodium cyanide injected into one of its veins, twenty-six cases were selected. In ten of these (Table II, *A*), the antecubital-to-carotid time was determined in the dorsal decubitus with the arm: (1) in the plane of the trunk, (2) elevated perpendicular to the trunk, and (3) dependent perpendicular to the trunk. When injections were made into elevated extremities, the needle was inserted into the vein with the extremity horizontal, the extremity then being elevated for one or two minutes with the needle and syringe in place, following which the injection was made.

In four individuals, similar determinations were made on the lower extremities (Table II, *B*).

TABLE II

EFFECT OF POSITION ON CIRCULATION TIME (SODIUM CYANIDE METHOD)*

A. Cubital Vein-to-Carotid Body Times With Upper Extremity Positioned (Patient Horizontal)

CASE	ARM HORIZONTAL	ARM ELEVATED	ARM DEPENDENT
1	12	12	15
2	18	14	20
3	17	17	27
4	17	18	22
5	15	17	15
6	12	11	16
7	11	13	16
8	10	10	14
9	14	15	20
Avg.	14	14	18

B. Ankle Vein-to-Carotid Body Times With Lower Extremity Positioned (Patient Horizontal)

CASE	EXTREMITY HORIZONTAL	EXTREMITY ELEVATED	EXTREMITY DEPENDENT
1	60	21	70
2	45	17	30
3	29	23	60
4	48	20	--

C. Ankle Vein-to-Carotid Body Times in Trendelenburg (Head Down, 30°) and Reverse Trendelenburg (Feet Down, 30°) Positions

CASE	HORIZONTAL	TRENDELENBURG	REVERSE TRENDELENBURG
1	26	25	31
2	35	20	42
3	35	25	66
4	43	18	150
5	over 2 minutes	22	no response
6	28	27	no response

D. Ankle Vein-to-Carotid and Cubital Vein-to-Carotid Body Times in Fowler's Position

CUBITAL-TO-CAROTID	ANKLE-TO-CAROTID (DORSAL DECUBITUS)	ANKLE-TO-CAROTID (FOWLER'S)
15	25	no response
13	28	no response
15	19	no response
17	20	no response
13	31	no response
12	20	no response

*Times in seconds.

In six cases a comparison was made between ankle vein-to-carotid times in the horizontal, Trendelenburg, 30° (head down, legs up), and reverse Trendelenburg, 30° (head up, legs down), positions.

In another group of six cases the ankle vein-to-carotid body time was determined with the patient in dorsal decubitus and in Fowler's position.

In two individuals, the effect of active movement of the foot and toes on the ankle vein-to-carotid body time in the dorsal decubitus position was determined.

RESULTS

From an inspection of Table I, it can be seen that, generally, one may expect to find a slightly shorter circulation time following such operations as are listed during the immediate postoperative period, especially following laparotomies. In the 191 cases listed, the average preoperative cubital-to-carotid and ankle-to-carotid times were seventeen and thirty-six seconds, respectively. In every individual, the cubital-to-carotid time was shorter than the ankle-to-carotid time. It should be noted that the circulation time varies between widely separated extremes in different patients. To obtain an idea of the individual case variations in circulation rate and the relation of these rates to the pulse and temperature at the time of determination, these data are included in Table I, *B*, which constitutes the largest case groups.

In the dorsal decubitus position, the antecubital-to-carotid time with the arm in the plane of the trunk averaged fourteen seconds; with the arm elevated perpendicular to the trunk, fourteen seconds; and with the arm dependent perpendicular to the trunk eighteen seconds (Table II, *A*).

In the dorsal decubitus position, the ankle-to-carotid time with the lower extremity at body level was sixty seconds, with the extremity dependent seventy seconds, and with the extremity elevated twenty-one seconds. In another individual, the horizontal time was forty-five seconds, but in the dependent position was reduced to thirty seconds; however, when elevated, the time was seventeen seconds. The values in the third and fourth cases are what might be predicted (Table II, *B*).

With the extremities in the plane of the trunk, the ankle vein-to-carotid time in six individuals was found to be, typically, thirty-five seconds with the trunk horizontal, twenty seconds in the Trendelenburg position, and forty-two seconds in the reverse Trendelenburg position (Table II, *C*).

In only one of six patients placed in Fowler's position could a response-to-ankle vein injection be elicited; in fact, in two cases, perivascular necrosis occurred at the point of injection, the venous pressure being so high in that position as to force the solution out the vein through the needle puncture. In each case, however, a response was noted when the patient was brought from Fowler's to the horizontal position (Table II, *D*).

In the dorsal decubitus position, active movement of the foot and toes in one individual decreased the ankle vein-to-carotid body time from thirty-two seconds in the resting state to thirty seconds in the active; in another individual the time was decreased from forty-five seconds in the resting state to twenty-one seconds in the active state.

DISCUSSION

The slight decrease in circulation time following operation may be due to the effect of the increased temperature and respiration on the pulse rate, a possibility entirely in accord with the observation that in none of the tabulated cases was there a great deal of postoperative abdominal distention.

The direct explanation of the slightly greater velocity of blood flow postoperatively is to be found in the cardiovascular system. Koch[14] has stated that the blood velocity is affected by the time of day, season, climate, heart force, stroke output, viscosity of the blood, pulse frequency, absorption of food, menses, pregnancy, age, and respiration. The most obvious explanation for the faster blood flow postoperatively is the elevation of body temperature with its corresponding increase in heart rate, Berns[2] having shown experimentally that artificial fever accelerates the blood flow in dogs. A fast blood circulation in fever was also observed by Wollheim and Lange[31] and Lian and Facquet.[17]

For a similar reason, blood flow is accelerated in thyrotoxicosis, and conversely, retarded in myxedema (Blumgart, Gargill, and Gilligan[3, 4]). Hering had insisted that cardiac rate did not influence circulation rate, and Steinhaus,[26] observing a slower pulse in the dog after vagotomy, concluded that circulation time was prolonged by increased heart rate. Tarr, Oppenheimer, and Sager[27] and Tigerstedt[29] have affirmed that circulation time has little or no relationship to heart rate or arterial blood pressure, but Vierordt showed that there was a positive correlation between pulse rate and circulation rate, a finding substantiated by Blumgart and Weiss[5] using radium C, who found, however, that a considerable increase in heart rate only increased the blood flow slightly.

Apparently, dependency of the upper extremity retards the return of its blood to the heart, although, conversely, a faster return in the elevated position could not be demonstrated. This is probably due to the fact that the emptied veins lack a sufficient medium in which the stimulant can be transported. The possibility that the Fowler's position tends toward venous stasis in the lower extremities should be seriously considered, especially in those conditions where thrombophlebitis is often an unhappy accompaniment. Frimann-Dahl[9] showed in four patients that raising the extremity increased the rate of venous flow. He, therefore, urged postoperative elevation of the lower extremities or the foot of the bed and frequent active movement of the legs.

Piorry[21] in his classical paper on posture and blood flow first called attention to the impediment to circulation in the standing still posi-

tion, proposing that syncope was due to encephalic anemia resulting from stasis below the heart. He further stated that arterial, venous, and capillary circulation were all dependent on position, and that it was gravity that caused veins to distend when the arm was dependent. Incidentally, it was he who called attention to the effect of gravity on the production of hypostatic pneumonia.

Since the brilliant researches of Piorry, confirming observations have appeared in large numbers. Among these are the investigations of Bock, Dill, and Edwards[6] and Thompson, Alper, and Thompson[28] who showed that the circulation rate is much longer (1.5 to 4.5 times) in the standing than in the recumbent position. Lawrence, Hurxthal, and Bock[15] observed a downward gradient of blood flow on going from the recumbent to the sitting to the standing positions. Specifically, in an athlete who had won eight of eighteen Marathon races, there was a reduction of 25 per cent in blood flow on going from the recumbent to the sitting position, and a reduction of 40 per cent in the standing position, the flow in the recumbent position being taken as the standard. By a different method, Rosen and White[23] came to the same general conclusions.

The effect of position on venous pressure is well known, von Recklinghausen[22] having observed a venous pressure in the foot equivalent to the hydrostatic difference between the foot and the heart. He concluded that the force of the heartbeat alone was insufficient to raise the venous blood from the feet to the heart in the vertical position. His original observations have been confirmed many times, Schott[24] having shown that the venous pressure in the foot is increased as the difference in the hydrostatic level between the foot and right atrium is increased. Hill and Barnard[11] have calculated that in a man six feet high the hydrostatic pressure of a column of blood from the vertex to the sole is about 140 mm. Hg, and from the vertex to the middle of the abdomen about 50 mm. Hg. It is, therefore, understandable why the Fowler's position retards the venous return from the lower extremities and pelvis.

Mogk[20] measuring directly the brachial venous pressure in dogs, found a 400 per cent increase after exercise. Burton-Opitz[8] found that in dogs venous flow from a limb was increased during muscular contraction and that the pressure also rose. Braune[7] described the mechanism by which the venous stream to the right heart from the lower extremities is accelerated by muscular movement, and later Kaufmann[13] demonstrated in the horse that muscular activity (mastication) causes blood to spurt from an open vein (of the masseter muscle). In the human being, Meinertz[19] found that the venous pressure in the hand turning a centrifuge was 1 to 8 cm. of water higher than in the inactive hand. Sewell[25] found that the venous pressure in the hand rose 3.4 to 5.3 mm. Hg on alternately clenching the fist, observations which have been confirmed by those of Hooker.[12]

SUMMARY

1. Usually, the cubital vein-to-carotid body time and ankle vein-to-carotid body time, as determined by the sodium cyanide method, is shortened after major surgery. In one-hundred and ninety-one cases, the average preoperative cubital vein-to-carotid body and ankle vein-to-carotid body times were seventeen and thirty-six seconds, respectively; postoperatively, the times were fourteen and thirty-two seconds, respectively.

2. Circulation rates vary widely in patients with apparently normal circulatory systems.

3. Acceleration of blood flow postoperatively is probably related to the increased metabolic demands of the organism, which are manifest by increased temperature, pulse rate, and respiration.

4. Gravity retards the flow of blood from the upper and lower extremities, either when the position of the extremity itself is changed or when the longitudinal axis of the entire body is altered.

5. Fowler's position delays the return of blood from the lower extremities.

6. Ankle vein-to-carotid body circulation rate is shortened by active motion of the foot or toes and by elevation of the extremity.

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SEQUELAE OF TRANSFIXATION OF BONE

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A CRITICAL search for the cause of undesirable sequelae of bone transfixation in fracture therapy leads one to conclude that they arise not from the use but from the misuse of transfixions. Further analysis reveals the misuse is usually due to fear, unpreparedness, or haste, with resultant failure to observe the following dictums of bone transfixation.

1. Proper placement of the transfixions
2. Fixation of the transfixions to their respective fragments
3. Control of forces available through transfixation

The most prolific source of complication is the improper placement of transfixions. Fear of violating a joint has erroneously led the "authorities" to advocate transfixing the shaft of bones. Such piercing of the shaft, still insisted upon by even the most recent textbooks, is fraught with danger. Not only are the shafts narrow but the cortical bone is so dense that it contains the minimum blood supply. When all the traction and countertraction pressure exerted by a pin is focused upon such a relatively small area of poorly vascularized bone, a pressure necrosis may result, much in the same manner that a sustained pressure on the big toe of a patient with Buerger's disease will precipitate a local gangrene. Why penetrate the shaft when the wide, richly vascular, honeycombed diaphysis can be more safely and quickly transfixed.

Placement of the transfixion in a bone beyond the distal fragment, such as the os calcis for tibial shaft fractures or through the crest of the tibia for femoral shaft fractures, is not infrequently productive of complications. Such placement in an adjacent bone, while it may supply sufficient traction, cannot prevent loss of reduction or movement between fragments, with the possibility of delayed union. However, when the transfixions are placed directly through the fragments, they supply not only traction and countertraction but positive fixation.

Insertion.—Surprising as it may seem, the actual process of fast drilling of a large pin through dense bone such as the shaft of the tibia will generate so much heat at the pin point that an actual local cauterization occurs. Such coagulation and burning of the haversian canals cause an *aseptic thermal necrosis.* Results of this lethal heating will be expressed, one or more months later, in the form of a doughnut-like or ring sequestrum.

While thermal necrosis usually results from improper placement or use of dull pin points, it may result from inserting pins or half-pins at excessive speeds with a hand drill or electric motor.

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Asepsis.—It seems unnecessary in our present stage of enlightenment to more than mention the need of strict asepsis, yet infractions of the rules still occur. Failure to mask both mouth and nose of everyone in the room and handling of the transfixions without a recently boiled hemostat may result in septic grief. Skin incisions for insertion of transfixions, use of medicated dressings, or redressing of pin wounds will only propagate further sequelae.

Late pin-wound infections are often produced by senile, youthful, or uncooperative patients who insist upon scratching the wound with

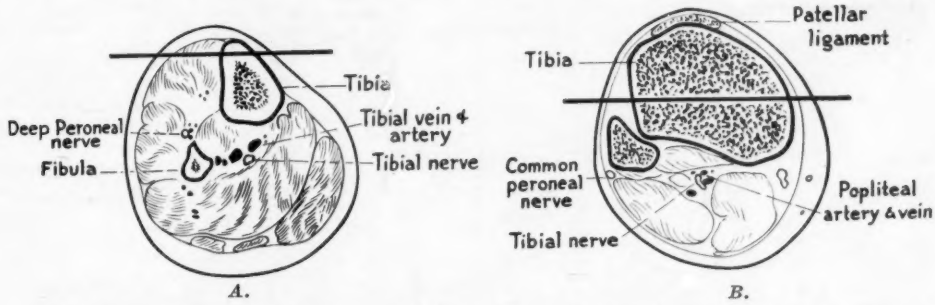


Fig. 1.—*A*, Improper placement of transfixions is the most prolific source of sequelae. For instance, piercing the crest of the tibia is physiologically and mechanically unsound. The bone is so dense that the friction of drilling generates enough heat to cause a local thermal coagulation with subsequent ring necrosis. Focusing all the skeletal traction force on a small avascular area frequently results in a pressure "sore." Drilling in such hard bone causes the pin to wobble with a resulting loose fit of pin in bone. Coupled with narrowness of the bone at this level, this makes for instability.

B, In this case the lazy way is the correct way. Insertion of the pin through the wide, cancellous bone, near but not into the joint, is easily done and supplies excellent control and immobilization. Traction pressure is distributed equally to many little partitions. Furthermore, the multiplicity of honeycombed cells minimizes subsequent sidewise movement. The vascularity assures immunity and early repair.

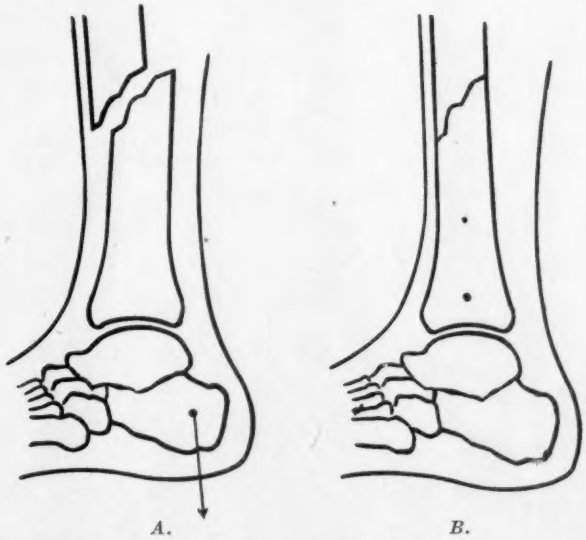
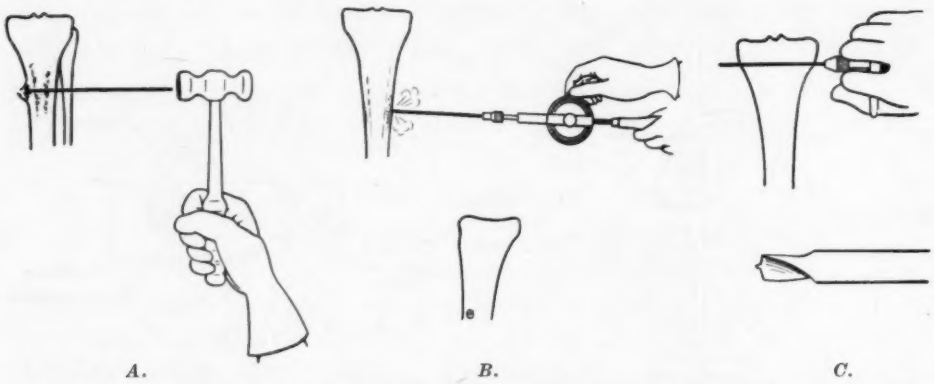


Fig. 2.—*A*, Skeletal transfixation of the os calcis supplies traction, but control of the distal tibial fragment is lost through mobility of the two intervening joints.

B, Transfixion of the fragment itself serves a triple purpose: (1) direct traction, (2) positive control, and (3) absolute fixation. Whenever it is necessary to transfix the cortical or shaft portion of the femur, a slender pin or wire is preferable, provided it is kept taut.

finger or stick. With this type of patient it is well to cover transfixion areas with large dressings or with a segment of plaster cast.

Immobilization of Transfixions.—Regardless of the location and care with which a wire or pin is inserted, it is evident that subsequent movement of the bone upon the transfixion can but result in a frictional irritation with discharge and pain. Breaks in the technique that result



A. *B.* *C.*

Fig. 3.—*A*, Why place a pin through bone that is so dense it is necessary to use a hammer? Such an act often further fractures the shaft and explodes the exit hole. A pin so placed is both painful and so loose that accurate reduction cannot be maintained.

B, The placement of pins through dense cortex, use of thick pins with dull points, or excessive haste will cause a thermal necrosis of surrounding bone with a late ring sequestrum.

C, Insertion of pins with special sharpened points at proper locations, and at a slow speed with hand drill or electric motor, avoids most, if not all, cases of aseptic thermal necrosis which have been erroneously termed infection.

Fig. 3*D*.—Improper placement of the pin through the crest of the tibia resulted in typical ring sequestrum. The eradication of this sequel required more time, expense, and pain than the original fracture.

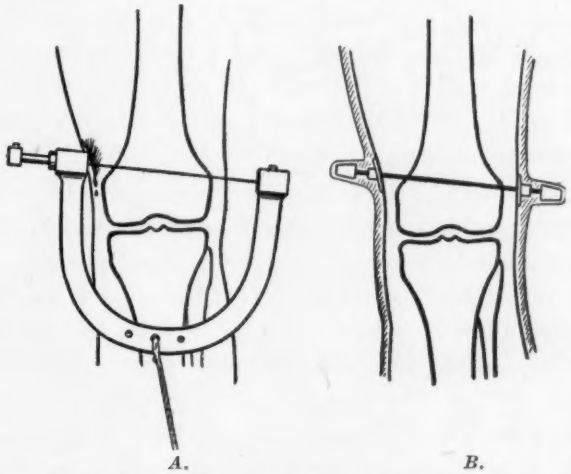
in movement between fragment and transfixion, with their share of sequelae, are noted below.

1. Failure to angularly insert two transfixions into each fragment, or to fix firmly the single transfixion or parallel transfixions into the fragment by some positive means, such as a snug-fitting cast.

A. *B.*

Fig. 4.—*A*, Sterility of transfixions should be assiduously preserved. The ordinary lifting forceps and bottle should be ostracized because it is invariably polluted after it is used a few times.

B, Covering of the nose as well as the mouth of everyone in the room is imperative. Placing of corks over both ends of the transfixion keeps the pin points sharp and prevents their falling through holes in the tray. Pins should not be touched with the gloved hand but only with a recently boiled hemostat.



A. *B.*

Fig. 5.—*A*, Failure to limit side motion between transfixion and bone is productive of complications.

B, A firm binding of the transfixion to the fragment is essential for success. Incorporation of the pin forestalls contamination from the patient's fingers, holds the reduction, eliminates infection from sidewise slipping, and avoids all pain. If location of fracture and associated wounds permits, it is better to insert angularly two transfixions into each fragment.

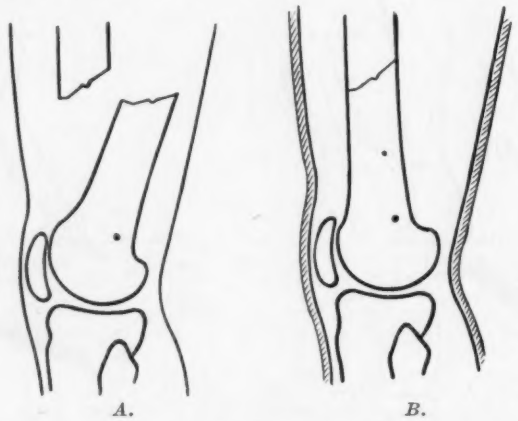
2. Employment of a lax wire through use of an inadequate horseshoe or reliance upon the sides of the cast to keep the flexible transfixion taut.

3. Improper placement of a transfixion through an adjacent bone rather than through the fragment itself.

4. Insistence on hammering the transfixion through the bone, in which event an enlarged opening is often "exploded" in the opposite cortex.

5. Needless fear of injuring deeper structures leads to the selection of too small a half-pin unit or to incomplete penetration of the opposite cortex with the half-pin.

6. Failure to employ an adequate number of transfixions is the error productive of most complications.



A. *B.*

Fig. 6.—*A*, It is impracticable to control the deformity in a supracondylar fracture by single transfixion as the fragment is free to pivot on a single pin.

B, Double transfixions supply means of "freezing" this fragment in one position throughout convalescence. Paradoxical as it may seem it is much safer to employ several than a single transfixion.

Paradoxical as it may seem, it is safer to use several rather than a single transfixion. The fragment encased in a cast can pivot or teeter-totter on a single transfixion, and as atrophy occurs and the swelling recedes the bone may slide sidewise in the cast; whereas, if two or more pins, wires, or half-pins are placed at an angle to each other, movement of the fragment on the transfixions is impossible.

It is an absolute necessity for half-pin transfixions to go through both cortices if subsequent movement is to be avoided; yet in the use of our half-pin unit a not infrequent cause of undesirable sequelae is failure to drill through the opposite cortex. Penetration of a single cortex not only fails to fasten the pins securely into the bone but subsequent movement of the bone on the pin will often cause pain, discharge, and loss of reduction.

Control of the Forces Available Through Transfixation.—For earliest possible union it is necessary for the fragments to be immobilized in direct contact during the entire healing process. Throughout the convalescence period absorption occurs at the ends of the fracture fragments. This is especially evident in transverse fractures in the distal

Fig. 7A.—If half-pins do not penetrate beyond the opposite cortex, movement with pain and loss of apposition will occur.

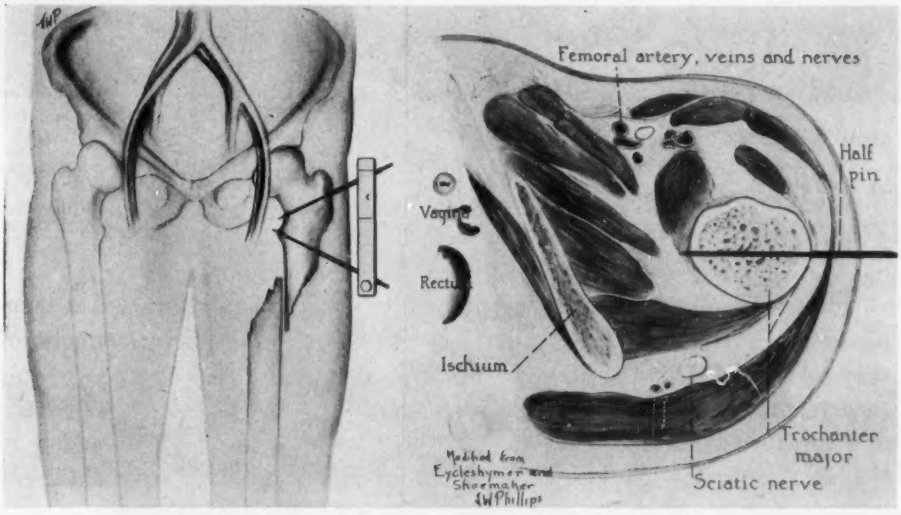


Fig. 7B.—Half-pins may penetrate safely the opposite cortex of any shaft fracture as every fragment has a practical and safe portal of entry.

one-third of the tibia. This bony absorption, which we attribute to trauma of the osseous-vascular system, is not visible for several weeks or months. Therefore, transfixions which at first hold the fragments in perfect apposition may be, in transverse fractures, the same means that will hold the fragments apart after absorption has taken place.

A. *B.*

Fig. 8.—*A*, The surest way of obtaining nonunion is to immobilize the fragments in distraction.

B, X-ray of the same patient eighteen months later when he first came under our care. In spite of a sliding bone graft, nonunion has persisted, partially accounted for by the distraction of a united fibula.

Before the days of modern fracture treatment, malunion was a common complication; but the thoughtless and enthusiastic employment of skeletal distraction has now reversed the trend and placed that greater catastrophy, nonunion, in the ascendency, well explained on the basis of overextension. However, when these same transfixions are used in the reverse manner (i.e., for "contaction") then and only then can unions be obtained in the shortest possible time.

Failure to keep a patient ambulatory, now feasible with our newer knowledge of transfixation, provokes a long train of undesirable sequelae such as stiff joints, muscle and bone atrophy, circulatory atresia, edema,

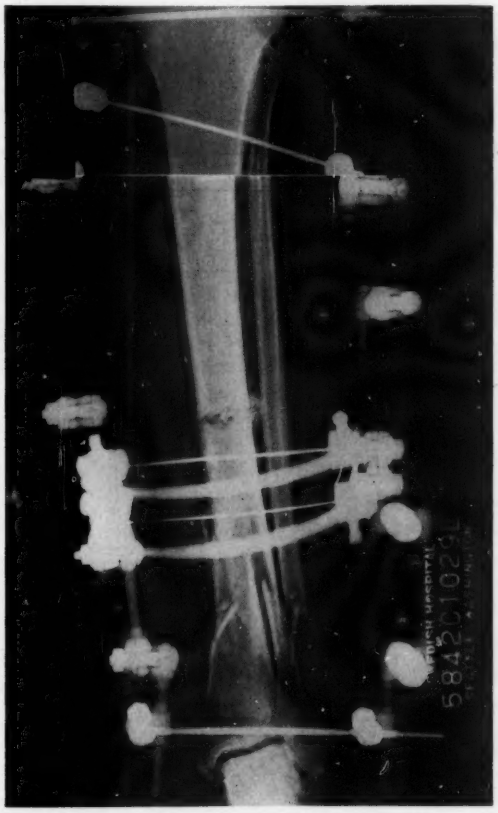


Fig. 9*A*.—The quickest method ever devised of obtaining union is by "contaction" procured through double transfixation of each fragment. In this print the films have been spliced together to show two transfixions in the upper fragment and two Kirschner wires in central fragment. There was room for only one pin in the distal fragment. The angle in which the x-ray was taken makes it appear that the pin was in the ankle joint.

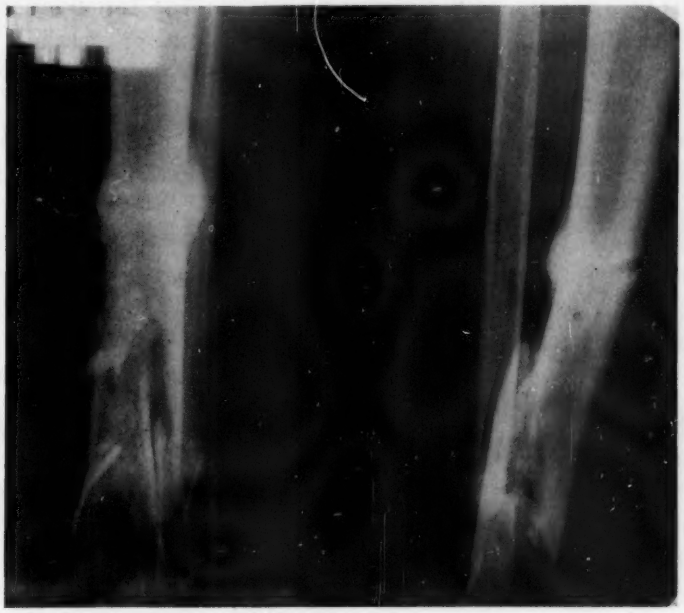


Fig. 9*B*.—Final result, four months later.

and general metabolic and psychic changes. In order that these be reduced to a minimum, it is highly desirable that ambulation and active joint movement be insisted upon. Improved vascular and lymphatic circulation, through function and muscular contraction, decreases the swelling, and ambulation prevents marked muscle and bone atrophy and joint stiffness. With an increased active blood and lymph flow it is axiomatic that more callus and union in less time is not only possible but probable.

Fig. 10.—A. I., aged 28 years, with fractured shaft of the femur. The psychologic, physiologic, and economic benefits of early ambulation with free joint movement are numerous.

Prophylaxis.—Complications, as a class, are avoidable and may be limited to a minimum approaching zero when the following technique is carefully employed. Insert transfixions, whenever possible, through the wide cancellous portion of the bone. Place two pins, two wires, or two half-pins at an angle to each other into each fragment. Use the transfixions for traction only in reduction; then reverse the procedure and use the transfixions, for a "contaction" means to keep the ends of the fragments in direct and immobilized contact throughout the whole period of repair. Employment of the above clinically tested principles, and insistence on ambulation with active muscular and joint movements, will be rewarded by an earlier bony union than can be attained by any other method.

RÉSUMÉ

Sequelae are usually due not to use but to misuse of transfixation. The common errors causing the majority of complications, arising from fear, unpreparedness, or haste, may be classified under (1) improper placement; (2) movement between fragment and transfixion; (3) use of transfixions for skeletal traction instead of contaction; and (4) failure to use transfixions so that the benefits of ambulation can be realized.

RESECTION OF FEMORAL NECK WITH PELVIC SUPPORT OSTEOTOMY FOR ANKYLOSIS OF THE HIP

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THE therapy of ankylosing affections of the hip has swayed between a natural desire to achieve painless stability and a laudable wish to preserve or restore painless mobility. The result is that the orthopedic surgeon has employed his utmost energies in perfecting methods for insuring firm fusion of the hip joint and then equally determinedly devised the most ingenious techniques for undoing the ankylosis which may present itself. This apparent inconsistency is readily comprehensible and would be completely justifiable were there any assurance of success, either in the arthrodesing or in the arthrolysing procedures. Such is not the case and it is this inadequacy which may possibly condone the following presentation.

Essentially, the method here suggested is a combination of two separate surgical procedures. The first, designed to preserve stability by making a new point of pelvic support other than the femoral head, is accomplished by means of the Lorenz bifurcation osteotomy. The second, directed toward the re-establishment of mobility, is accomplished by a modification of the pseudo-arthrosis principle recommended by Jones. As a matter of fact, the operative approach was a natural development of the treatment of the fractured neck of the femur. It is of considerable interest to recall that the bifurcation operation suggested by Lorenz was originally the result of his observations of a war-incurred fracture of the upper end of the femur. Subsequently, the bifurcation was successfully employed routinely in the treatment of nonunited fractures of the femora. It was as a consequence of the satisfactory results in the care of this condition that Dr. H. Finkelstein suggested the conversion of the ankylosed hip into a pseudo-arthrosis, which could then be stabilized by means of the pelvic support osteotomy.

The first of these operations was performed in 1934. Since that time some seven or eight other hips have been similarly treated. The results have not been uniformly good. But in certain types of cases where the need for mobilization of the ankylosed hip has been urgent, and where the advisability of a formal arthroplasty was questionable, the operation has proved of value. This is illustrated in the case here reported.

C. K., aged 10 years, first came to the Hospital for Joint Diseases, March, 1928, with the complaint of pain and swelling in the left knee joint. These symptoms appeared following an injury some two months before. The mother stated that at the age of twenty-six months the child had been treated at the Ruptured and Crippled Hospital for a left hip limp, of nine months' duration. An abscess of the left

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thigh was incised and drained. Traction was applied and subsequently the thigh was immobilized in a plaster of Paris spica. No definite bacteriologic diagnosis was established, though tuberculosis was undoubtedly suspected, since the immobilization was continued for a period of four years. Upon removal of the plaster, the symptoms had disappeared, but the child was left with a marked limp, due to ankylosis of the hip in adduction and flexion.

On admission to the hospital, the left hip was fixed in 10 degrees of adduction and about 30 degrees of flexion. There was no active motion, but the suspicion of a very slight amount of external rotation on passive motion presented. The roentgenogram was reported as showing a destructive arthritis of the hip joint, with bony ankylosis. The knee joint was palpably the site of a marked effusion. Extension of the leg was limited. Roentgenogram of the knee joint showed no bony changes. The Wassermann test was negative, but the intradermal tuberculin test was positive in all dilutions. Fluid aspirated from the knee was sterile on ordinary culture. (Guinea pig innoculation was subsequently reported to be negative.) A diagnosis of nonspecific synovitis of the knee was made, as previously it had been made elsewhere, with respect to the hip. The knee was immobilized in a plaster of Paris mould. In May, 1928, the child was discharged to the outpatient department.

Under conservative treatment, the acute manifestations subsided. The child remained relatively free of symptoms until May, 1931, when she was again admitted to the hospital for pain and swelling of the knee. At this time it was noted that slight rotation was present in the hip and doubt as to the nature of the ankylosis was expressed. The roentgenogram (Fig. 1) was reported as showing an old ankylosing process of the left hip. However, later restudy disclosed that the head and neck were absorbed, that the joint space was completely obliterated, but that below the femoral head, in the region of the epiphyseal line, there was an irregular zone of radiotranslucency, similar to that which has since been seen in cases of epiphyseal pseudo-arthrosis.[1]

Though the specific therapy of this condition was not at that time recognized, a pelvic support osteotomy was performed, but for other reasons. Because of the necessity for immobilization of the knee, it was decided to correct the hip deformity at the same time and so save the patient unnecessary hospitalization. On May 15, 1931, a subtrochanteric osteotomy was attempted. The distal fragment, however, became displaced medially, so that in effect a Lorenz bifurcation was performed. Specimen of the bone removed at the time of operation showed no evidence of any tuberculosis. The convalescence was uneventful, and in June the patient was discharged in a plaster of Paris spica. In September, the plaster was removed and the patient was permitted to bear weight on the affected limb. Physiotherapy was instituted as soon as the patient's pain subsided.

Following her discharge from the hospital, the patient made intermittent visits to the dispensary. In 1936, roentgenogram revealed a destructive lesion in the external condyle of the left femur, which was considered to be tuberculosis. The pain gradually became worse, so that in October, 1937, the patient could no longer bear weight upon the knee joint. She was immobilized in plaster and advised bed rest, but without relief of symptoms. In November, hospitalization was advised. At this time the knee was found swollen, tender, and warm to the touch. There was a moderate valgus, which was attributed partly to the tuberculosis and partly to the antecedent bifurcation operation. Motion at the knee was limited to 155-degree extension and 100-degree flexion. The hip was solidly fused and painless (Fig. 2).

The patient now presented an extremely disturbing problem. She was 19 years of age and was suffering from what was clinically tuberculosis of the left knee. This had been treated conservatively for a period of about nine years, but without

success. The indication for surgical fusion of the knee joint appeared to be urgent. On the other hand, the already existing fusion of the hip joint seemed to present an equally formal contraindication to a second fusion in the same limb. The only solution to the dilemma lay in a release of the hip before proceeding with ankylosis of the knee.

Fig. 1.—Roentgenogram (C. K.) showing that the joint space has been obliterated, but the remnants of the head are still to be seen. In the region of the epiphyseal line, there is a zone of radiotranslucency.

At that time the bifurcation resection operation was being developed and it was determined to employ this in preference to a more standard type of arthroplasty. This was a purely fortuitous, but entirely fortunate decision, as the event proved. On Nov. 12, 1937, the hip joint was approached through an anterior Smith-Petersen incision. The ankylosis was released by an osteotomy performed on the neck, close to and parallel with the pelvic wall. No effort was made to gouge the head out of the acetabulum. This released the femur, but to prevent the possibility of re-ankylosis, the whole of the neck down to its base was excised in a single piece. No tissue was interposed and the wound was closed in layers. The knee was immobilized in plaster.

As a matter of routine, the specimen was submitted to histologic examination. Microscopic section disclosed "the spongy trabeculae to be atrophic and the cortex thin. Almost at the center of the section is a caseous, partly calcified mass. It is surrounded by a collar of epithelioid cells and collagenous connective cells. About

this is a zone of lymphocytes and plasma cells, in which collections of epithelioid and giant cells may be observed.

"*Diagnosis.*—Tuberculoma of the neck of the femur, in a case of old, healed tuberculosis of the hip joint."

Motion of the hip was started ten days after operation and walking with the aid of crutches was permitted at the end of six weeks. The patient left the hospital on Jan. 1, 1938. In July, 1938, the range of motion at the hip included flexion from 170 to 145 degrees, external rotation 5 degrees, adduction 15 degrees from the midposition, and abduction 5 degrees from the midposition. Actively, the patient was able to raise the leg from the examining table, even though she was wearing a Thomas walking brace to protect the knee.

Fig. 2.—The hip is solidly fused; the characteristics of the Lorenz bifurcation have disappeared and the femur presents the appearance of a Schanz type of pelvic support osteotomy.

Examination in April, 1940, revealed essentially the same arcs of passive motion, without any increase in active power. The knee joint was flexed at 145 degrees and roentgenogram of the knee joint disclosed destruction of the lateral femoral condyle. "There is very little motion at the knee and the process appears to be slowly progressing." Admission to the hospital for fusion of the knee was recommended, but was refused by the patient, because of the subsidence of acute symptoms. The patient was seen again in May, 1941. The conditions in the hip were essentially unchanged. The patient was able to get about with moderate comfort

and could sit down. Even though she had but little active power in the hip muscles and walked with a limp, she had a remarkably stable and painless hip. Roentgenogram of the hip taken at this time (Fig. 3) was reported as showing "a firmly united bifurcation osteotomy of the femur, with the upper end of the distal fragment forming a new joint between it and the base of the ascending ramus of the pubic bone close to the acetabular ring."

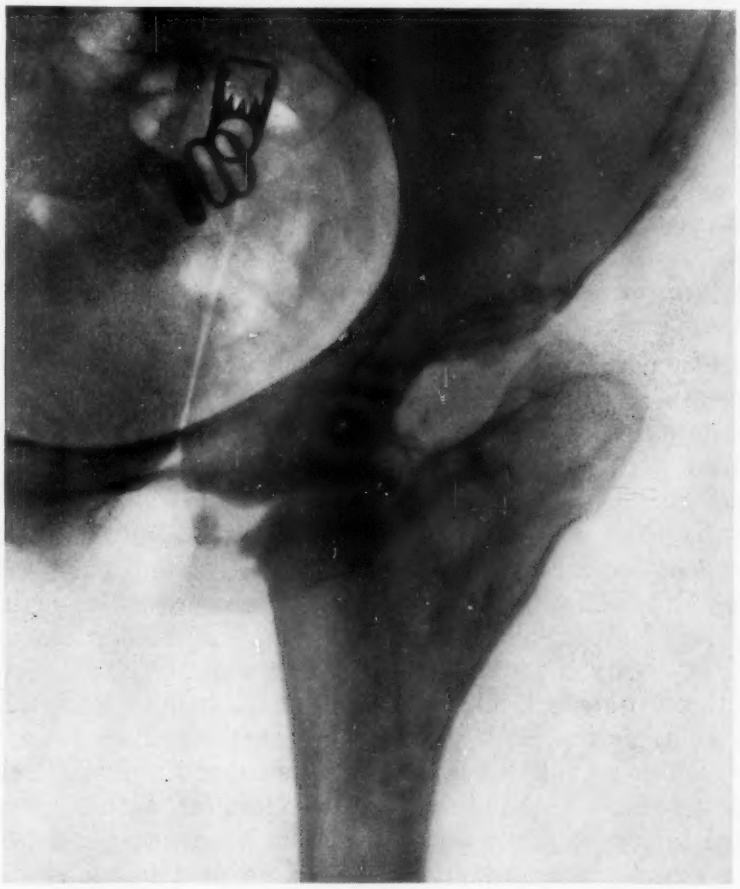

Fig. 3.—The femoral neck has been resected and motion restored. Stability is maintained by pelvic abutment of the apex of the postosteotomy angle.

This position is of course not ideal for the re-establishment of physiologic motion. In fact, it may be one of the factors responsible for the inability actively to move the limb. On the other hand, the limitation of active motion may be due to intrinsic loss of power in the muscles themselves. All too frequently sight is lost of the fact that mere reconstruction of the mechanical conditions for motion does not assure the result, unless some means of restoration of the motor power, that is of the re-establishment of the biomechanics of the joint, is guaranteed. In a patient such as has been described here, where passive motion far exceeded the range of active motion, it must be presumed that the muscle motors had been damaged beyond repair by the antecedent inflammatory

process and by the subsequent prolonged immobilization during the period of fusion. This objection applies to all types of arthrolysing operations, quite irrespective of the mechanical efficiency of the joint which may be the result of the surgical intervention.

Whether the surgical effort in any particular case should be directed toward the remodeling of a new anatomic hip joint, or merely toward release of the ankylosis, will be largely a question of individual preference or experience. The startling discovery of a tuberculoma of the neck in the case here reported suggests the possibility of a disastrous reactivation of the process, had the usual type of arthroplasty been performed. On the other hand, the uneventful response following resection of the neck would appear to speak in favor of the procedure in these types of cases. It would seem to have its special field of usefulness in those cases in which the absence of a femoral neck would preclude the performance of the usual type of arthroplasty.

A further advantage of the operation is that it is performed in two stages, neither of which is shocking to the patient. The operation has been performed at one sitting, but this is considered inadvisable, since the plaster immobilization necessitated by the osteotomy prevents the mobilization which is desirable after release of the ankylosis. At the outset, the effort was made to mobilize the hip before attempting osteotomy of the shaft. While this materially reduces the technical obstacles, some difficulty was experienced in the prevention of upward displacement of the shaft between the first and second stages. At present, osteotomy is performed as the first stage. This may be done simply through a small, lateral incision, at the base of the trochanter, or may be performed with greater precision through an anterolateral incision, which permits accurate displacement of the distal fragment. Following abduction of the osteotomized fragment, both hips are immobilized in plaster, until firm bony union has taken place. In performing the abduction, no attention need be paid to the size of the postosteotomy angle.[2] Since the head and neck are to be removed, the pelvis will be supported by only a single fulcrum at which no limitation of motion can occur. Nevertheless, excessive abduction is to be avoided and 30 degrees may be taken as an average maximum of abduction for the distal fragment.

Following re-establishment of bony continuity at the site of the pelvic support osteotomy, the second stage for release of the ankylosed head is undertaken. If the head can be easily separated from the acetabulum, it may be exenterated and removed in one piece with the neck, which should be cut through at its base. If, however, the ankylosis is so solid that the head cannot be easily delimited, no effort should be made to gouge out the acetabulum, after osteotomy at the base of the neck. The femoral head should be released by another osteotomy made flush with the pelvic wall. Traction and graded active and passive motion should

be instituted as after any other arthrolysis. But, contrary to the practice in other arthroplastics, weight bearing may be permitted as soon as the postoperative reaction has subsided.

SUMMARY

An operative procedure for the treatment of hip ankylosis is presented. In principle, the operation consists of two separate operations performed in successive steps: the first stage, the Lorenz bifurcation osteotomy, undertaken in order to insure stability of the hip; the second stage, release of the hip by resection of the femoral neck or head, is performed when bony union following the osteotomy has occurred. This combination affords the opportunity of satisfying both primary requirements of hip function, stability and mobility. It can be employed in cases where the classical types of arthroplasty cannot be employed. The operation is relatively free of shock, and can be carried out at intervals that require relatively short periods of enforced hospitalization. Each of the steps in the operation is simple and does necessitate the refinements of organization that formal arthroplasty makes essential.

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DOUBLE PULLEY HUMERAL ADAPTATION OF RUSSELL TRACTION

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IN TREATING fractures of the shaft of the humerus many methods are available, depending upon the type of fracture, its location, associated injuries, and the surgeon's experience and ability. In the hands of an expert the frequent election of open reduction may produce gratifying results, whereas its general use may be disappointing. The immediate application of a cast, with or without some form of ambulatory traction, has its place in selected cases but frequently a better immediate and final result with a shorter ultimate period of incapacity and, as Colp and Findlay[1] have shown, a quicker economic restoration may follow hospitalization with extension.

There are many indications for caution in the employment of the bedside extension methods. Speed[2] has recently emphasized the fact that the most frequent error committed is "overpull" with resulting interposition of soft parts or periosteum, preventing reapposition and frequently resulting in delayed or nonunion. Inadequate traction or loss of traction, through errors in application of the equipment or arrangement of pulleys, and inadequate nursing care may cause overriding or malalignment.

Certain complications common to the various methods of application may also occur. The use of too much weight may cause adhesive to slip and injure the skin, resulting in infections which interfere with subsequent operations.

As Heyl[3] has pointed out, skeletal application of traction permits the use of greater weights and skin injuries are avoided. It must be remembered, however, that every wire, nail, or tong causes a compound injury to the bone and carries with it the hazards associated with that type of injury. Mathewson[4] calls attention to the fact that incorrect angle, avulsion, bending or slipping of the wire or nail, injury to nerves and blood vessels, pressure necrosis or gangrene of soft parts, hematoma formation, and infection of the skin or bone are occasionally encountered when skeletal traction is employed.

The system most commonly used in applying traction to the humerus consists of two or three separate weight and pulley combinations as presented in 1918, by Blake and Bulkey,[5] one pulling through an overhead pulley on a Balkan frame to a hand spreader, another through a pulley on a traction stand for lateral traction of the humerus, and a third, through a pulley on the Balkan frame to a sling under the humerus.

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This multiple weight system requires considerable care in its application, constant observation, and frequent adjustments. A further disadvantage is the inability of the patient to change position in bed. The necessity of maintaining a flat supine position for about six weeks or until some bony union has resulted creates a definite nursing problem and causes the patient a great deal of discomfort.

Certain modifications of pulley arrangement such as that described by Blum[6] have simplified and improved this system of traction but without simplifying the nursing problems presented by the bedridden patient.

Herewith, we present a new and simple method for the application of traction which will permit the patient to assume a lying or sitting position in bed, thus simplifying the nursing care. The following is not a description of a new apparatus, but of a physiologically correct axis traction adaptation for use in selected fractures of the shaft of the humerus in which hospitalization is indicated.

ADAPTATION

The patient is placed in bed with the emergency Thomas splint in place and the traction is applied without altering the position of fragments as revealed by roentgenogram upon admission.

A Balkan frame is placed on the bed and a single pulley is attached to an overhead extension arm which is adjusted to allow the desired degree of abduction and flexion. The usual type of hand cage or spreader is applied by adhesive strips to the dorsal and volar surfaces of the forearm. A spreader with one double or two single pulleys attached is fastened to the antecubital region by a felt sling or by moleskin or adhesive tape. In the latter case the tender medial aspect of the arm is avoided. The adhesive strip is attached posteriorly and is passed outward over the spreader where it is divided and faced, each half passing about the forearm without adhering until, as it encircles the arm, it reaches the tape posteriorly.

The rope attached to the hand spreader is passed through the pulley on the overhead extension arm of the Balkan frame in order to support the forearm. From there it is passed through one-half of the double pulley system at the elbow, then through a single pulley attached to the end of the Thomas splint and, finally, through the other pulley at the elbow down to the weight below. In this way lateral traction and countertraction are produced to maintain humeral extension.

The pulley arrangement is shown in Fig. 1. In Fig. 2 is shown a superimposed parallelogram of forces with *OA* extending from the elbow toward the single overhead pulley. This force equals that of the suspended weight. The force *OB*, from the elbow through the double pulley system laterally, equals twice the suspended weight. When deducting the weight of the forearm and hand cage it is found that about three-fourths of the suspending force *OA* has been neutralized leaving *OA′* as the force entering into the parallelogram of forces act-

ing at the elbow. The resultant *OX*, slightly greater than *OB*, is found to lie in the axis of the shaft of the humerus.

A three-pound weight exerting its traction through the single overhead pulley will produce a three-pound lift to support the arm and hardware, and through the double pulley system, it will produce slightly more than a six-pound extension traction upon the fracture site.

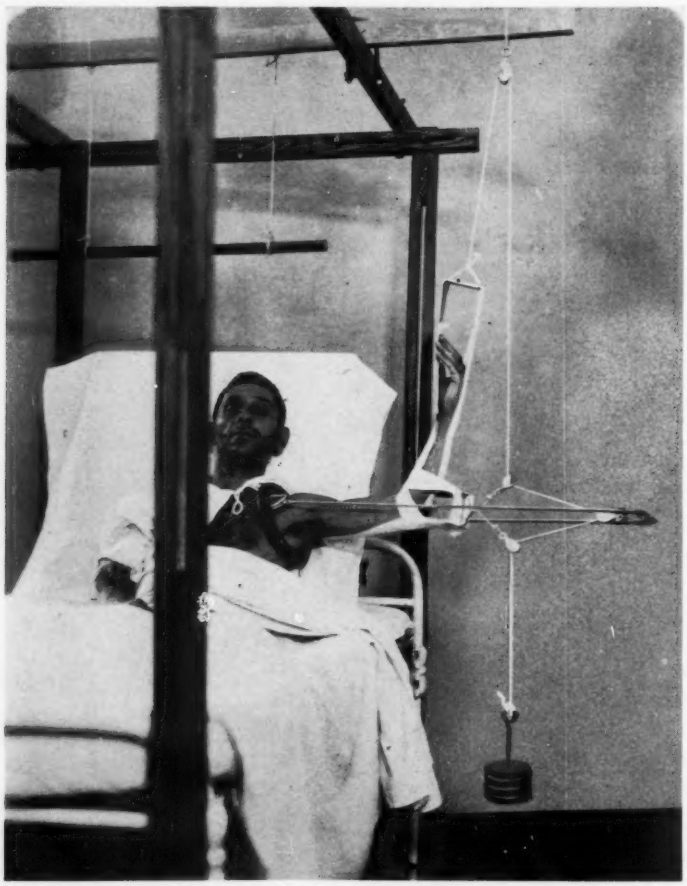

Fig. 1.—Double pulley traction method of humeral extension in fractures of the shaft of the humerus, showing application and pulley arrangement. Bandage and humeral sling are omitted for purpose of illustration. Lowering of back rest will allow patient and apparatus to descend. The rope travels through the pulley system as the weight is raised.

It has been found that arms requiring more or less traction also require a correspondingly heavier or lighter weight for their support. By using weights from two to six pounds this system is, therefore, applicable to children as well as to muscular adults. To decrease or increase the lift on the forearm the overhead pulley may be shifted outward or inward, thereby increasing or diminishing the resultant forces involved in lateral traction. A much simpler method, however, is to lock the overhead pulley in the desired position by means of a plug or clamp.

A muslin sling may be fastened over the bars of the Thomas splint to support the humerus. It may become necessary to support the splint with tape applied to the forearm or with rope attached to or above the hand spreader.

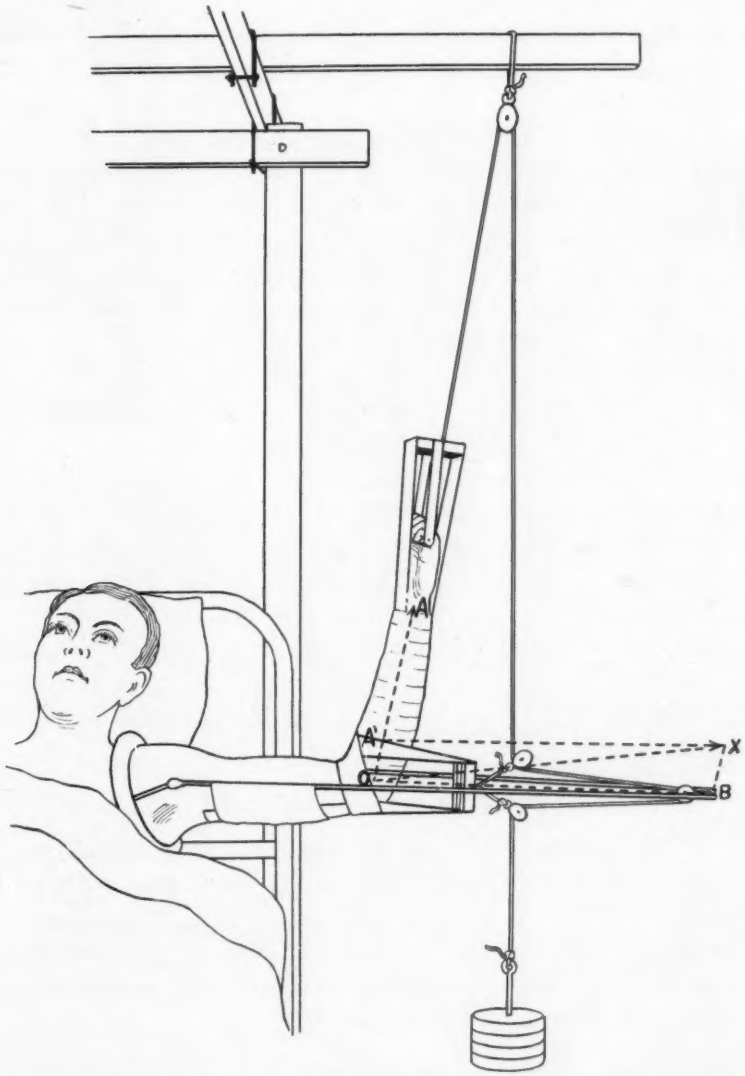

Fig. 2.—Illustration showing superimposed parallelogram of forces. *OA*, Equals that of the suspended weight; *OB*, acting through the double pulley system, equals twice the suspended weight. *OA'* is the portion of *OA* entering into the parallelogram of forces (*A'A* being neutralized by the weight of the arm and hand cage). *OX*, the resultant of forces *OA'* and *OB*, lies in the axis of the humerus.

When the adjustments are all made, the outstanding advantage of this system over other methods of extension becomes apparent. As the patient rises on the back rest, the nurse gently lifts the end of the Thomas splint and the rope travels through the system of pulleys as the weight descends. Merely lifting the weight allows the arm and splint to descend as the back rest is again lowered.

ADVANTAGES

1. No special equipment is required.

2. It is comfortable and it may be adjusted with ease; no angle calculations are necessary.

3. No tilting of the bed is required; there is no loss of traction caused by the elbow spreader striking the pulleys as there may be with the usual traction stand.

4. Less supervision is required by the nursing staff.

5. There is no posterior angulation of the fracture when the patient is raised from the mattress for the usual nursing care; the sling attached to the Thomas splint rises with the extension system.

6. The patient may be readily transferred from Thomas splint emergency traction; he may be safely transported to the x-ray department for studies when portable equipment is not available.

7. This system is adapted to skeletal traction by means of a wire through the distal humerus or olecranon, or to modifications of skin traction as advocated by Blum[6] and Butcher.[7]

8. There is less force exerted upon the arm for corresponding amount of axis traction than in other forms of multiple pulley systems, such as that described by Blum,[6] as there are no widely separated opposing forces producing a resultant. The single pulley force overhead is practically equalized by the weight of the arm and hardware, while the greater, double pulley force corresponds with the desired axis for humeral traction.

9. The freedom of motion afforded makes the patient's hospitalization much more bearable.

SUMMARY AND CONCLUSIONS

Some of the common methods of management of fractures of the distal half of the shaft of the humerus are mentioned with comments on the deficiencies of the existing methods of bedside traction systems.

A new double pulley method of humeral extension is described which eliminates many of these deficiencies and provides certain advantages which other extension methods do not offer.

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INTESTINAL ACTIVITY FOLLOWING DISTENTION OF THE GALL BLADDER AND URINARY TRACT: AN EXPERIMENTAL STUDY*

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THE gastrointestinal manifestations accompanying diseases of the biliary tract or of the urinary tract are well known. In many cases these diseases are accompanied by some transitory or permanent gastrointestinal symptom, that is, nausea, vomiting, epigastric distress, abdominal discomfort, ileus, distention, constipation, and so forth. These secondary symptoms are at times so conspicuous as to cloud the true nature of the primary disturbance and to lead to an incorrect diagnosis. A possible explanation of some of the symptoms noted clinically in these cases may be found in recent experimental work in this field of research. To date this has dealt mainly with the stomach but we shall concern ourselves only with the effect of distention of the gall bladder or of the urinary tract on the small intestine.

Schrager and Ivy found that when the gall bladder of the dog was distended by inflating a toy balloon placed in its lumen, the animal experienced discomfort and occasionally salivation and vomiting. Distention of the biliary ducts caused marked discomfort; pressures of more than 100 mm. of mercury always produced salivation and frequently produced vomiting. It was evident that the symptoms were more intense and uniform when the biliary ducts were distended than when the gall bladder was distended, and the symptoms could be elicited with less pressure.

Zollinger performed similar studies on man. The gall bladder and the common duct were distended after the patient had recovered sufficiently from a short period of anesthesia to answer questions intelligently. Distention of the gall bladder did not cause vomiting, whereas distention of the common duct frequently did cause vomiting.

Welch, Coplan, and Holmes stated that roentgenoscopic observations on the duodenum and jejunum of patients who had pathologic changes in the ureter indicated that there are spasm of the duodenal cap and hyperperistalsis of the descending loop of the duodenum, sometimes so severe as to cause regurgitation.

King inserted a cannula through the apex of the bladder of barbitalized dogs, so that the bladder could be filled or emptied at will by raising or lowering a pressure bottle connected to the cannula in the bladder. Intestinal records were made with a balloon-tambour system.

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He found that when the pressure bottle was raised very slowly, no change of duodenal activity took place. The response to sudden and rapid increase of pressure was somewhat variable, but quantitative rather than qualitative. King never observed any augmentation of duodenal tonus and movements under these conditions, but quite constantly he observed a diminution. In many of his cases the relaxation was small, in others it was prompt and complete.

The objective of the investigation reported here was to determine if changes in the activity of the jejunum and ileum could be produced by distention of (1) the gall bladder, (2) the upper part of the urinary tract, or (3) the urinary bladder.

METHOD OF STUDY

Female dogs, weighing between 8 and 17 kg., were used in these experiments. All the animals were trained for making the necessary observations and recordings. All operative procedures were made with the animal under ether anesthesia, using surgical technique. Studies were not done until healing had occurred. Advantage was taken of the technique devised in this laboratory[3] for obtaining samples of intestinal contents and used subsequently for the study of intestinal peristalsis.[7] This preparation consists of a loop of small intestine in continuity with the remainder of the bowel, with blood and nerve supply intact, enclosed in a bipedicled loop of skin. Records of intestinal activity were obtained by means of a double tambour system, the receiving tambour being fastened to the loops of intestine by an aluminum clip, and the recording tambour being attached to a recording lever, the excursions of which were recorded on the kymograph.

Distention of the Gall Bladder.—Distention of the gall bladder was accomplished by inflating a fine rubber balloon which had been inserted through the fundus into the lumen of the gall bladder. The balloon had been tied previously over the end of a size 16 self-retaining catheter in such a way that when it was filled without tension it was of approximately the same size as the cavity of the gall bladder. The opening in the fundus was closed securely around the catheter, which was brought to the outside through a stab wound. The gall bladder was distended by injection of saline solution at the body temperature of the animal (39 to 40° C.) from a syringe into the catheter-balloon system in the gall bladder. The catheter was fitted with a glass T tube, to provide for the inclusion in the system of a simple mercury manometer. In this way the gall bladder could be distended slowly or rapidly to whatever pressure was desired and this pressure could be maintained for whatever period was desired. The distention was maintained usually for fifty to sixty seconds and an interval of several minutes elapsed between successive trials.

The experiment was first done on the animals recently fed and was repeated after a twenty-four- to forty-eight-hour fast. In this manner, the effect of distention of the gall bladder was studied in all phases of

intestinal activity, from the vigorous, steady activity in the recently fed animal to the infrequent, feeble, and irregular contractions which interrupt long periods of quiescence in the fasted animal.

Distention of the Upper Part of the Urinary Tract.—Distention of the upper part of the urinary tract was accomplished by injecting water or dilute urine through a ureteral catheter. The details of technique in the procedure of ureteral catheterization have been worked out by Greene and his method was used. The animal was anesthetized with pentothal sodium administered intravenously. A 5 per cent solution was injected at a rate of about 1 c.c. per minute. From 4 to 6 c.c., depending on the size of the animal, were necessary to induce a stage of anesthesia suitable for work with the cystoscope. The animal was maintained at this level by the injection of 0.5 to 1 c.c. of the anesthetic agent from time to time as necessary. Intravenously administered pentothal sodium is very satisfactory for this work, because the animal wakes within one-half hour after administration of the anesthetic agent has been discontinued and completely recovers within two hours. There is very little vomiting or other gastrointestinal disturbance, and the animal will eat shortly after waking.

With the animal under anesthesia as described, the urethra was dilated to a caliber of 28 French with van Buren sounds. A 24 French Braasch cystoscope then could be introduced readily into the bladder. The ureteral orifices were identified and a suitably sized ureteral catheter (5 or 6 French) was manipulated through the ureterovesical valve of the more easily accessible ureteral orifice and passed on up into the pelvis of the kidney. The cystoscope then was removed, the catheter being left in place and fixed with adhesive tape to the perineum. An attempt was made throughout the procedure to maintain sterile technique.

The presence of the catheter in the ureter did not disturb the animal to any discernible degree, and once the animal had recovered completely from the anesthetic, it appeared normal in every respect. The animal was fed twenty to thirty minutes before the start of the experiment to insure active peristalsis during the study.

With the trained animal tied on the padded frame, water or human urine at 39° C. was injected from a syringe into the ureteral catheter. In several of the experiments, the catheter was pulled down 8 to 10 cm. after studies had been made with the tip of the catheter in the renal pelvis, and the experiment was repeated with the tip of the catheter in this lower position. The length of the ureter in the dog as determined from post-mortem studies varies according to the size of the animal; in animals of the size studied (12 to 16 kg. respectively) the ureter was found to be about 22 to 25 cm. in length.

Each subsequent catheterization was performed on the same animal after an interval of two to three weeks. In this interval of time between catheterizations, the trauma and edema resulting from the procedure had subsided and the bladder and ureteral orifices, as seen through the

TABLE I

DISTENTION OF UPPER PART OF THE URINARY TRACT:
DOG 1, LOOP OF JEJUNUM 10 TO 12 INCHES (25 TO 30 CM.) FROM LIGAMENT OF TREITZ (WEIGHT, 14 KG.)

DATE	SIZE URETERAL CATHETER (FRENCH)	CATHETER IN RIGHT OR LEFT URETER	POSITION OF CATHETER TIP	FLUID INJECTED (C.C.)	DURATION OF INJECTION (SEC.)	DURATION OF INHIBITION (SEC.)
3/14/40	7	L	Pelvis	8	15	20
				8	20	25
				10	20	25
				10	15	25
				10	40	60
				10	20	35
				10	30	70
3/29/40	6	L	Pelvis	6	40	180
				5	60	240
			8 cm. below pelvis	5	60	300
				8	35	50
				10	60	150
				5	60	90
				6	60	150
				6	30	50*
				7	20	20
			13 cm. below pelvis	6	20	30
				9	40	75
4/22/40	5	R	Pelvis	10	150	150
			8 to 10 cm. below pelvis	20	210	210
				40	300	300
				15	300	0
				10	25	25
				50	120	120
				10	40	90

*Vomited.

cystoscope, appeared normal in every respect. From seven to eleven determinations were made at five-minute intervals on each occasion that the ureteral catheter had been passed.

Distention of the Urinary Bladder.—This study was performed under the following subdivisions: (a) rapid distention, (b) slow distention, (c) maintenance of inhibition, and (d) cystometric studies.

In order to fill the bladder, a two-way Robinson latex catheter of suitable size was passed into the bladder and fixed in position with adhesive tape. Water or dilute urine at the body temperature of the animal (39 to 40° C.) was run into the bladder from a 500 c.c. graduate type of flask elevated about 90 cm. above the level of the body of the dog. A Murphy drip unit provided for regulation of the flow to any desired rate. Intravesical pressures, when studied, were determined by connecting a simple water manometer to the second passage of the catheter.

RESULTS

Distention of the Gall Bladder.—Determinations were made on three animals, one with an exteriorized loop of jejunum taken about twelve inches (30 cm.) from the ligament of Treitz, and two with exteriorized

TABLE II

DISTENTION OF UPPER PART OF THE URINARY TRACT:
DOG 2, LOOP OF ILEUM 10 TO 12 INCHES (25 TO 30 CM.) FROM CECUM*

DATE	POSITION OF CATHETER TIP	FLUID INJECTED (C.C.)	DURATION OF INJECTION (SEC.)	DURATION OF INHIBITION (SEC.)
3/14/40	Pelvis	6	35	60
		6	20	180
		6	20	150
		6	20	40
		6	20	90
		6	20	60
		6	20	100
		6	20	120
		6	20	90
3/29/40	Pelvis	10	50	50
		10	50	50
		10	25	25
		10	40	70
	10 cm. below pelvis	10	20	20
		10	40	40
		10	30	30
		10	25	30
4/9/40	Pelvis	6	35	65
		10	50	150
		10	50	70
		10	35	55
	10 cm. below pelvis	6	35	180
		10	50	60
		10	50	78
		8	40	120

*Weight 12 kg., catheter (5 French) in left ureter.

loops of ileum, taken about twelve inches (30 cm.) from the cecum. Twenty-four determinations on different days (eight for each of the three animals) were made on the recently fed animals. In no instance did distention of the gall bladder in the pressure range of 0 to 400 mm. of mercury produce any effect on peristalsis, as observed directly from the loop or recorded on the kymographic record. There was no acceleration or inhibition of the peristaltic waves and no change in tonus, and the essential pattern of the kymographic record was unchanged.

Fifteen determinations on different days (five for each of the three animals) were made on the animals after they had been fasted for twenty-four to forty-eight hours. In these instances the bowel was in a relatively quiescent state with only occasional peristaltic activity. Distention of the gall bladder through the pressure range of 0 to 400 mm. of mercury failed in each instance to stimulate the quiescent bowel.

In 38 per cent of the instances of distention of the gall bladder to pressures of more than 140 mm. of mercury, salivation was noted and in all instances pressures of more than this value produced discomfort. Vomiting did not occur.

Distention of the Upper Part of the Urinary Tract.—Distention of the upper part of the urinary tract produced inhibition of activity in both the jejunal and ileal loops (Tables I and II). For the most part, the

inhibition persisted only as long as the duration of the injection of the fluid; however, in some instances inhibition persisted for as long as five minutes after the fluid had been injected (Figs. 1 and 2). The animals experienced some discomfort while the upper part of the urinary tract was distended. Salivation occurred quite generally during the periods of distention and on one occasion (Dog 1) vomiting occurred.

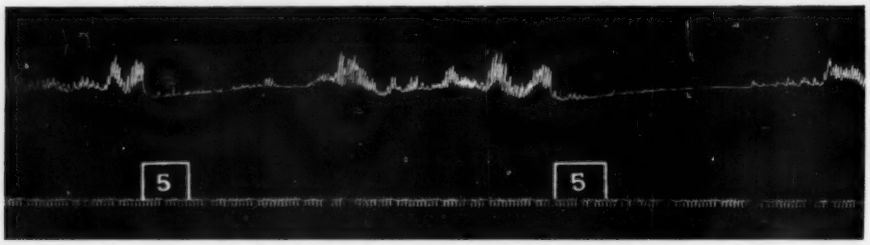

Fig. 1.—The upper part of the left ureter was distended with 5 c.c. of saline solution injected through the ureteral catheter over a period of fifty-five seconds in the first instance depicted. Inhibition of motility of a jejunal loop developed immediately and was maintained for four minutes. In the second instance depicted, 5 c.c. of saline solution were injected over a period of fifty-five seconds; inhibition developed immediately and was maintained for six minutes. The time intervals represent five seconds and one minute.

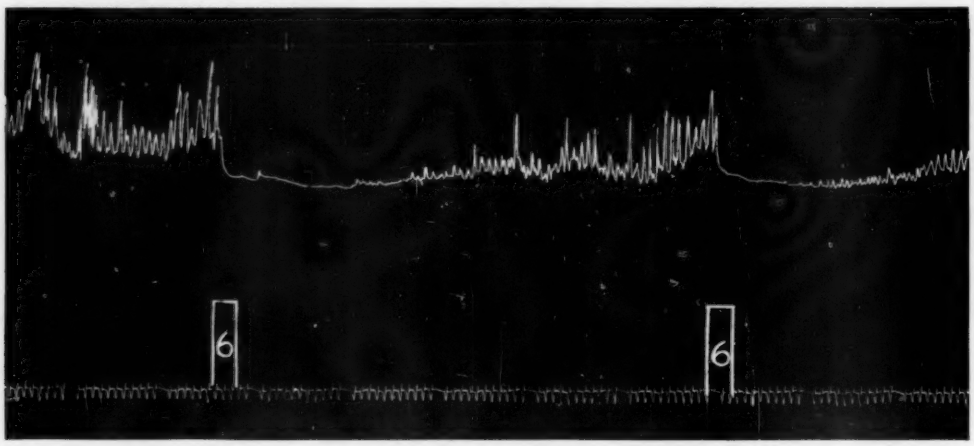

Fig. 2.—In the first instance depicted, 6 c.c. of saline solution were injected through the ureteral catheter into the upper part of the left ureter over a period of twenty seconds. Inhibition of motility of an ileal loop developed immediately and persisted for three minutes. In the second instance depicted, 6 c.c. of urine were injected over a period of twenty seconds; inhibition of motility developed immediately and persisted for two minutes. The time intervals represent five seconds and one minute.

Distention of the Urinary Bladder.—Studies were made on four dogs with exteriorized loops of the lower part of the ileum, ten to twelve inches (25 to 30 cm.) from the cecum, and on three dogs with exteriorized loops of the upper part of the jejunum, ten to twelve inches distal to the ligament of Treitz. The animals were fed fifteen to thirty minutes before the experiment was started to insure intestinal activity.

Rapid Distention: Rapid distention was performed eighty-four times on seven animals (twelve times on each dog). With a rate of flow of 100 c.c. in twenty to eighty-five seconds, it was found consistently that inhibition of activity occurred (Figs. 3 and 4). In some instances in-

hibition began when as little as 20 to 50 c.c. of fluid were present in the bladder, while in other instances 200 to 250 c.c. were necessary to produce the effect. The amount of fluid in the bladder necessary to suppress motility varied from animal to animal and in the same animal from day to day. Usually, however, the effect could be elicited when less than 100 c.c. of fluid had been introduced rapidly into the bladder.

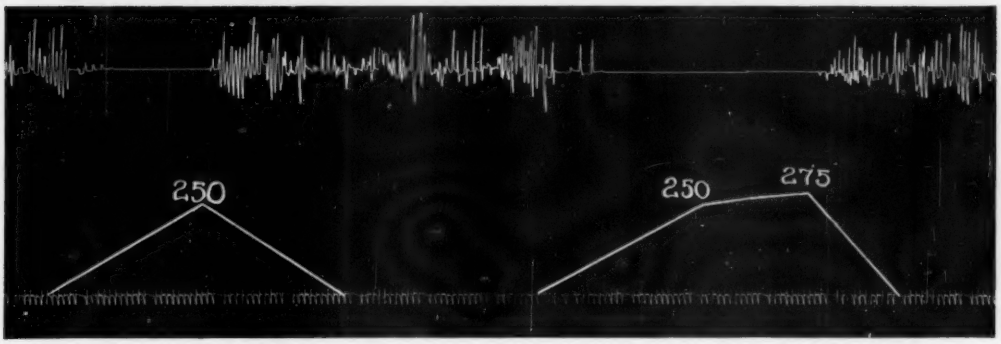

Fig. 3.—Loop of jejunum 10 to 12 inches (25 to 30 cm.) from ligament of Treitz. Bladder filled at rate of 100 c.c. in fifty seconds. Inhibition of intestinal activity occurred when 30 c.c. had been introduced into the bladder. Intestinal activity was resumed immediately after the catheter was opened. Time intervals represent five seconds and one minute.

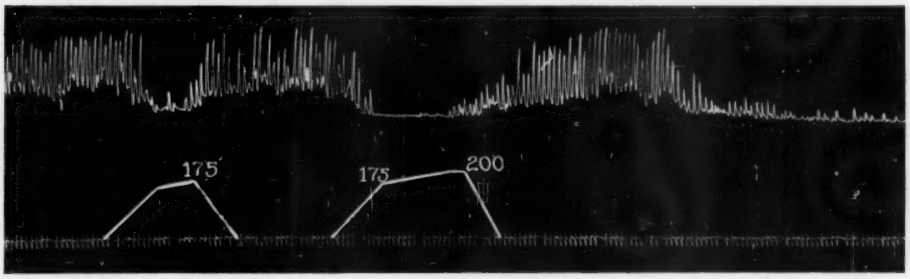

Fig. 4.—Bladder filled at rate of 100 c.c. in thirty-five seconds. Inhibition of activity of an ileal loop resulted when 100 c.c. had been introduced in the first instance depicted, and in the second instance, when 150 c.c. had been introduced. Resumption of intestinal activity occurred immediately after the catheter was opened. Time intervals represent five seconds and one minute.

Intestinal activity was resumed immediately after the catheter was opened and the fluid permitted to escape from the bladder.

Slow Distention: The urinary bladder is filled normally from the ureters at a rate of about 2 c.c. per minute. This rate of filling of the bladder was found to be too slow for animal experimentation, because the animal became fatigued and restless before the bladder could be overdistended at this rate. McLellan, in cystometric studies on human subjects, found that the optimal rate of filling of the bladder for determination of cystometric pressures was 5 c.c. per minute. After several preliminary determinations in which the element of fatigue of the animal was taken into consideration, it became apparent to us that the slowest rate of filling of the bladder suitable for animal experimentation by our method was of the magnitude of 7 to 14 c.c. per min-

TABLE III

SLOW DISTENTION OF THE URINARY BLADDER OF DOGS WITH EXTERIORIZED LOOP OF LOWER PART OF ILEUM

NUMBER AND WEIGHT OF DOG	RATE OF FLOW (C.C. PER MIN.)	CYSTOMETRIC PRESSURE AT ONSET OF INHIBITION (CM. OF WATER)	BLADDER VOLUME AT ONSET OF INHIBITION (C.C.)	FINAL BLADDER VOLUME (C.C.)	DURATION OF INHIBITION (MIN.)	CYSTOMETRIC PRESSURE WHEN URINATION OCCURRED (CM. OF WATER)
1	8	–	350	465	30	– –
14 kg.	8	–	340	430	18	– –
	7	18	375	440	50	42-46
	7	27	380	435	62	46-50
	8	13	400	500	25	48-54
Average		19.3	369	454	37	45-50
2	7	18	360	375	29	46-48
13.5 kg.	7	20	325	350	21	52-56
	7	17	360	460	44	44-60
	8	35	400	410	10	50-54
Average		22.5	361.2	398.7	26	48-54
3	7	27	330	355	38	50-56
12 kg.	8	20	280	380	36	40-44
	14	22	280	280	10	50-54
	14	26	325	345	38	44-48
Average		23.8	303.8	340	30.5	46-50.5
4	7	30	210	250	29	40-48
8.5 kg.	14	35	130	200	76	44-50
	14	33	70	100	16	54-60
	8	35	325	360	23	56-60
Average		33.2	183.8	227.5	36	48.5-54.5
Average of averages		24.7	304.4	355	32.3	46.9-52.2

ute. Therefore, in the experiments designated "slow distention" the bladder was filled at rates ranging between these limits.

Seventeen determinations were made on the four animals with ileal loops. Inhibition of intestinal activity occurred in every instance. However, a considerably larger volume of fluid in the bladder was necessary to initiate this reflex than when the bladder was distended rapidly. The range of volume of fluid in the bladder when inhibition occurred in these studies was 70 to 400 c.c., the average of the series being 304.4 c.c. It is to be borne in mind that these animals varied in size, and it is rational to suppose that the capacity of the bladder varies in direct proportion to the size of the animal. This assumption may serve, in part, to explain the wide range of volume of fluid in the bladder necessary to initiate the inhibitory reflex in the various animals. Thus, the animal in which 70 c.c. of fluid in the bladder produced inhibition of activity weighed 8.5 kg. (Dog 4, Table III), while the animal in which 400 c.c. in the bladder were necessary to produce the phenomenon weighed 14 kg. While it was found to be generally true that the smaller the animal the less fluid in the bladder was required to produce reflex inhibition of intestinal activity, it was also found that variations in this respect occurred in the same animal from time to time.

TABLE IV

SLOW DISTENTION OF THE URINARY BLADDER IN DOGS WITH EXTERIORIZED LOOP OF UPPER PART OF JEJUNUM

NUMBER AND WEIGHT OF DOG	RATE OF FLOW (C.C. PER MIN.)	CYSTOMETRIC PRESSURE AT ONSET OF INHIBITION (CM. OF WATER)	BLADDER VOLUME AT ONSET OF INHIBITION (C.C.)	FINAL BLADDER VOLUME (C.C.)	DURATION OF INHIBITION (MIN.)	CYSTOMETRIC PRESSURE WHEN URINATION OCCURRED (CM. OF WATER)
5	8	–	–	380	–	50-56
16 kg.	12	–	–	550	–	48-54
	12	30	150	220	10	42-48
	12	–	–	430	–	46-50
	8	34	260	350	28	44-50
	8	–	–	400	–	48-52
Average		32	205	388.3	19	46-52
6	8	18	180	265	26	42-50
8.5 kg.	8	22	200	275	23	44-48
	14	20	260	320	7	45-58
	14	20	50	100	16	56-60
	8	17	190	260	12	50-54
	14	18	220	275	13	46-52
Average		19	183	249.1	16.1	47-54
7	10	–	–	410	–	56-60
12.5 kg.	11	–	–	390	–	48-52
	14	–	–	470	–	50-54
	7	19	410	450	16	42-46
	8	–	–	475	–	42-46
	10	–	–	500	–	46-52
Average		19	410	449.1	16	47-52
Average of averages		23	266.1	362	17.03	46.6-52.6

An interesting variation in response to slow distention of the bladder was noted in the animals with exteriorized loops of jejunum (Table IV). Three animals with such loops were studied in eighteen experiments. Whereas in the animals with loops of the lower part of the ileum inhibition was produced in every instance by slow distention of the bladder, in the animals with jejunal loops this phenomenon occurred in only nine of eighteen experiments, or in 50 per cent of the instances. This reflex was initiated on only one occasion in six attempts in one dog, twice in six attempts in another dog, and in all six instances in the third dog. The range of bladder volume when inhibition occurred was 50 to 410 c.c., the average volume being 266.6 c.c.

Maintenance of Inhibition: Once inhibition of activity of the small intestine was established by sustained distention of the urinary bladder, it persisted for a variable time, after which feeble, irregular peristaltic contractions began to occur. If at this point the volume of fluid in the bladder was enlarged by a relatively small increment, inhibition was re-established for a time, after which activity would again begin feebly. A state of quiescence could be re-established by again adding to the bladder volume by a small increment. This cycle of events could be repeated until the animal urinated or became so uncomfortable that the experiment was terminated.

Thus, in a study of Dog 1 (lower ileal loop) the bladder was filled at a rate of 7 c.c. per minute. Inhibition occurred when the bladder volume was 380 c.c. Quiescence of the small intestine persisted in a complete degree for twenty-nine minutes and in a relative degree for six minutes more. After this thirty-five-minute period of inhibition activity was resumed. Bladder volume then was increased by 30 c.c. (to 410) at the same rate of flow, and complete inhibition developed and persisted for five minutes, being followed by feeble, irregular con-

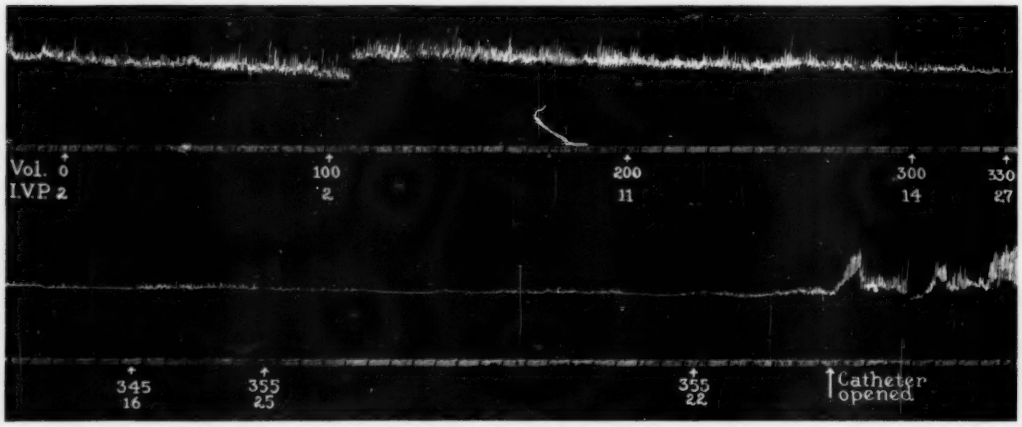

Fig. 5.—The bladder was distended at a rate of 7 c.c. per minute. When the volume of fluid in the bladder reached 330 c.c. inhibition of activity of an ileal loop occurred and persisted in a complete degree for six minutes and in a relative degree for six minutes longer. During this period of inhibition, the bladder volume had been increased at a uniform rate to 345 c.c. Activity then resumed and 10 c.c. were added to the bladder volume. This increment produced inhibition which persisted for twenty minutes in a complete degree and for six minutes longer in a relative degree. In all, then, inhibition was maintained in this instance for thirty-eight minutes. The experiment was terminated at this point because the animal experienced considerable discomfort. Time intervals represent five seconds and one minute. Vol.—volume in cubic centimeters; I.V.P.—intravesical pressure in centimeters of water.

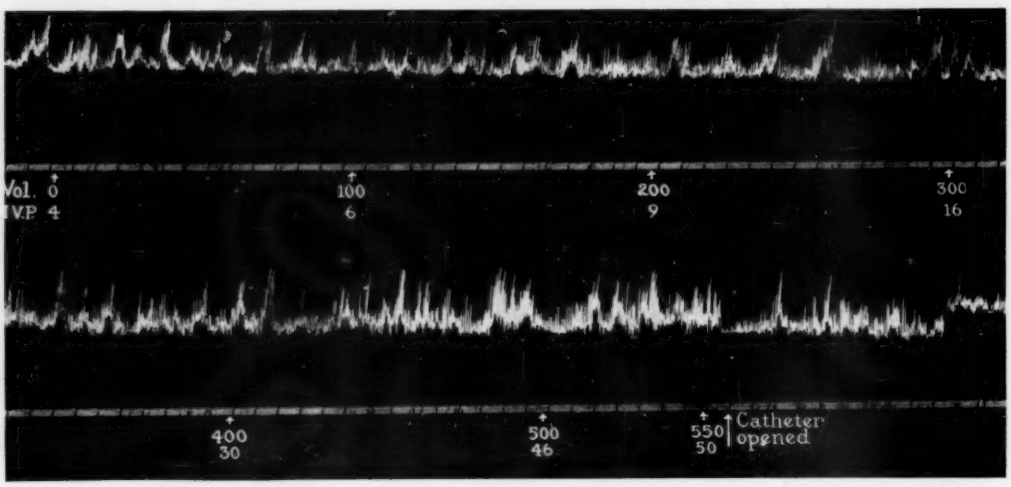

Fig. 6.—Loop of jejunum 10 to 12 inches (25 to 30 cm.) from the ligament of Treitz. No inhibition of activity resulted in this instance although the bladder was filled to 550 c.c. and the intravesical pressure rose to 50 cm. of water. Vol.—volume in cubic centimeters; I.V.P.—intravesical pressure in centimeters of water. Time intervals represent five seconds and one minute.

tractions (relative inhibition) for ten minutes longer. At this point activity again resumed. After 25 c.c. of fluid had been added at the same rate of flow, bringing the bladder volume up to 435 c.c. relative inhibition again began, persisting for a seven-minute period followed by a five-minute period of relative inhibition. At this point the animal urinated around the catheter. Thus, by adding small increments to the bladder volume from time to time, inhibition in this instance was maintained for sixty-two minutes. Another instance of the maintenance of inhibition of intestinal activity is illustrated in Fig. 5.

Seventeen experiments were performed on the four animals with lower ileal loops in an attempt to determine the length of time inhibition could be maintained by distending the bladder slowly. The average duration of inhibition obtained, as described previously, was 32.3 minutes, the range being ten to seventy-six minutes.

Here, again, a variance between the animals with jejunal loops and those with loops of the lower part of the ileum was noted. In three animals of the former group, slow distention of the bladder was per-

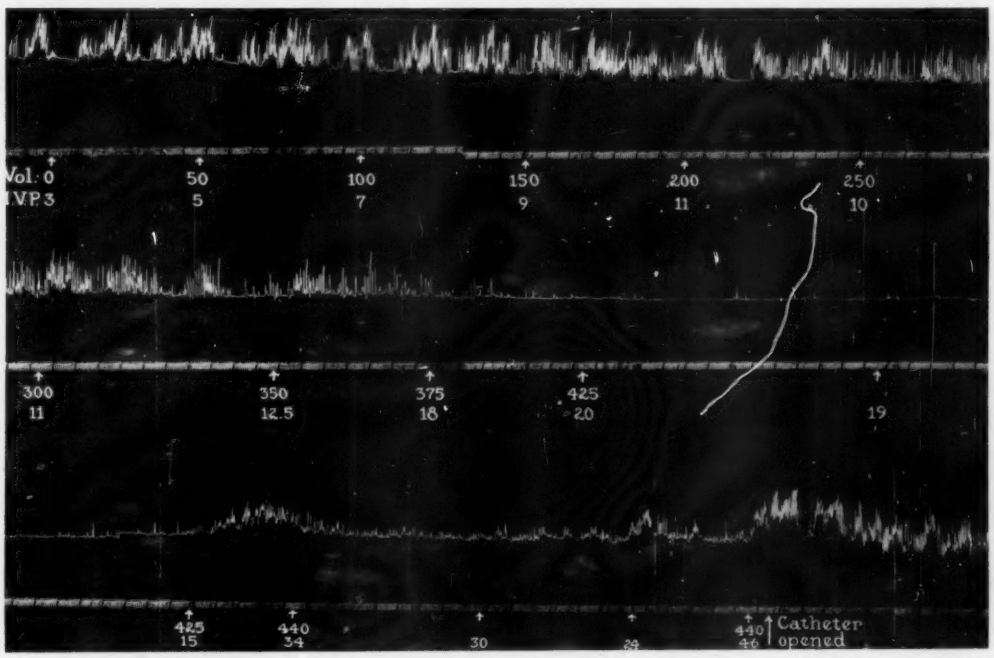

Fig. 7.—Fluid was introduced into the bladder at a rate of 7 c.c. per minute. The intravesical pressure gradually rose from 3 to 18 cm. of water, at which time inhibition of activity occurred with a volume of 375 c.c. in the bladder. After thirty-four minutes of inhibition (during the first seven minutes of which the bladder volume had been increased to 425 c.c.) the intravesical pressure had fallen to 15 cm. of water, at which time activity became re-established in the loop. The bladder volume then was increased by 15 c.c. (425 to 440 c.c.) in four minutes, after which intravesical pressure rose to 34 cm. of water, and a relative inhibition was re-established. The bowel remained relatively quiescent for sixteen minutes, during which time the intravesical pressure varied between 30 and 34 cm. of water. The animal was obviously uncomfortable at this point and was evidently attempting to urinate. As a result, the intravesical pressure rose sharply to 46 cm. of water, and the animal urinated around the catheter, after which intestinal activity immediately became vigorous and steady. Time intervals represent five seconds and one minute. Vol.—volume in cubic centimeters; I.V.P.—intravesical pressure in centimeters of water.

formed eighteen times. In nine instances no inhibition resulted (Fig. 6). In the nine instances in which inhibition did result, the average duration was 17.03 minutes, the range being seven to twenty-eight minutes.

Cystometric Studies: From the foregoing studies, one may theorize as follows: When the bladder wall is stretched sufficiently by a volume of contained fluid, impulses apparently arise which reflexly inhibit motility in the small intestine, particularly in the lower part of the ileum. As the wall of the bladder slowly accommodates to the volume of contained fluid, the tension in the wall is lessened and the inhibiting impulses are no longer generated. Motility therefore resumes; when the bladder wall is again stretched, by the addition of more fluid, inhibitory impulses are again sent out. Cystometric studies of intravesical pressures lend credence to this assumption.

It was found that, at the beginning of an experiment, when the bladder was empty, the intravesical pressure was of the order of 0 to 6 or 8 cm. of water. Inhibition of intestinal activity occurred at an average intravesical pressure of 23 cm. of water, the range being 11 to 35 cm. When intestinal activity re-established itself, the intravesical pressure usually had diminished to a value of 16 to 18 cm. of water. Urination occurred when the intravesical pressure reached an average value of 49.5 cm., the range being 46.6 to 52.2 cm. of water (Fig. 7).

COMMENT

One might be led to anticipate, from the knowledge of the work of Brush and Patterson, and of Fishback and Ivy, in which rather noticeable effects on gastric motility resulted from distention of the gall bladder, that the motility of the small intestine might be altered by distention of the gall bladder. However, no change in the peristaltic pattern of the jejunum or ileum, either in the recently fed or the fasted animal, was produced in any instance, even though the pressure in the gall bladder was raised to values far in excess of those employed by Brush and Patterson. The facts that, when pressures in excess of 140 mm. of mercury were applied to the balloon within the gall bladder, salivation occurred in 38 per cent of our cases, and considerable discomfort resulted in all instances, lead us to believe that in these experiments the gall bladder was being distended effectively. We can only conclude, therefore, that by our method of investigation, distention of the gall bladder through a pressure range of from 20 to 400 mm. of mercury does not produce any change in the activity of either the upper jejunal segment or the lower ileal segment of the small intestine.

Distention of the upper part of the urinary tract produced by the method employed in these studies persists only as long as fluid is being injected through the catheter. One could determine this objectively by noting that no fluid could be aspirated from the catheter at any time

during the injection or immediately following the completion of the injection. The inability to withdraw fluid through the catheter would lead one to believe that the injected fluid produced only a very transient distention and then rapidly leaked down around the catheter into the bladder. Several attempts were made to block the ureter at the ureterovesical orifice by passing a ureteral catheter fitted with a suitably placed wax plug. However, owing to the inherent difficulties of passing a ureteral catheter in the dog, this method was discarded because the results were not often enough satisfactory to make it feasible.

From this study, we have shown that distention of the upper part of the urinary tract of dogs produces an inhibition of activity of the jejunum and the ileum which persists in most instances as long as the distention is maintained, and in some instances for variable periods up to five minutes after the distention of the upper part of the urinary tract has been released. These results are in accord with the findings of Campbell and Patterson, of Welch, Coplan, and Holmes, and of Coplan, who found similar inhibiting effects on gastric and colonic activity from distention of the upper part of the urinary tract. These studies offer a possible explanation for the reflex gastrointestinal symptoms noted clinically in cases in which there are proved disturbances of the upper part of the urinary tract.

From our findings, and from the results of King, who found that sudden distention of the urinary bladder produced diminution of duodenal activity, certain generalizations may be drawn: (1) Sudden distention of the urinary bladder produces inhibition of activity in all segments of the small intestine. (2) When the bladder is distended slowly, certain variations of the response of the various segments of the small intestine are noted. King found that no change of duodenal activity occurred when the bladder was distended slowly, and in our experiments, inhibition of jejunal activity occurred in only 50 per cent of instances. These results in the upper reaches of the small intestine stand out in marked contrast to the consistent and prolonged inhibition resulting in the terminal portion of the ileum from slow distention of the urinary bladder.

Two logical conclusions may be deduced from the foregoing: (1) Sudden distention of the urinary bladder gives rise to a stronger inhibitory intestinal reflex than does slow distention. (2) Activity in the upper segments of the small intestine is less readily inhibited than is activity in the lower reaches of the small intestine by distention of the urinary bladder.

It has been the observation of many physiologists that the duodenum and jejunum are more active and more responsive to stimuli than are the lower reaches of the small intestine. Houckgeest, Flöel, Biederman, Lüderitz, and Bayliss and Starling all mentioned this observation in their writings. Hosoi and Alvarez, as stated by the latter,[1] by holding a short segment of bowel under the bath of a salt solution and against

an electrode, were able to show a graded response from duodenum caudalward to electrical stimulation. Alvarez also noted a remarkable gradient in the tendency of excised segments of rabbit's intestines to resume rhythmic activity after the segments had been thrown into spasm with pilocarpine and then relaxed again with atropine. Alvarez has also shown that there is a gradient from a short latent period in the lower part of the duodenum to a long one in the ileum, a gradient of tonus, a gradient of irritability, and a gradient of rhythmicity from the duodenum caudalward. Puestow later confirmed Alvarez' gradient hypothesis of rhythmicity in the dog.

Our results are consistent with the hypothesis of a gradient of irritability from duodenum caudalward. Since the upper segments of the small intestine are more irritable than are lower segments, it would seem that the duodenum and the upper part of the jejunum are less readily inhibited and less readily kept under inhibition by a given stimulus than is the lower part of the ileum.

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INTESTINAL ACTIVITY AFTER OBSTRUCTION OF COMMON BILE DUCT

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MANY physiologic alterations occur in obstructive jaundice. Some of the more important physiologic disturbances incident to obstruction of the biliary outflow occur in the gastrointestinal tract and considerable data have accumulated dealing with various gastrointestinal mechanisms, such as gastric secretion, intestinal absorption, and so forth, in the state of obstructive jaundice. The motor mechanism of the gastrointestinal tract in the presence of jaundice has not been thoroughly studied. Berti and Bernucci made a roentgenologic study of the small intestine in dogs after ligation of the common bile duct and found a delay in the passage of the opaque meal through the intestine. Gutzeit and Kuhlbaum conducted a similar study on a group of patients who had obstructive jaundice and on dogs after ligation of the common bile duct. Most of their patients had an incomplete obstruction of the duct. In their patients they found a decrease of intestinal activity that appeared to be due to depression of tonus contraction. In the dogs that had occluded bile ducts, the opaque meal reached the large intestine one or two hours later than in the normal state.

The purpose of our investigation was to study intestinal activity before and after ligation of the common bile duct using methods which permitted observation and recording of activity of loops of intestine under as nearly normal conditions as we have been able to devise.

EXPERIMENTAL PROCEDURES

This study was made on trained dogs that had exteriorized loops of jejunum and ileum. The exteriorized loops of intestines were in continuity, with intact mesenteric blood and nerve supply and enclosed in bipedicle tubes of skin. All operative procedures were made with the animal under ether anesthesia and with aseptic technique. The recording of intestinal activity was done by use of an air-displacement system which consisted of a rubber balloon attached around the loop by means of a rubber cuff and in communication through a rubber tube with a recording tambour.[3, 4]

All the animals were trained to lie quietly throughout the period of observation. Each period of observation was preceded by an eighteen- to twenty-four-hour fast and continued for various lengths of time after feeding on the table. A standard meal, consisting of milk, syrup, and

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bread, was given. This standard meal was used because it usually produces a characteristic motor response in the intestine of a fasted animal and the ingredients of the meal have been found most suitable for maintaining animals that have obstruction of the biliary outflow.

After sufficient control observations had been made, the animal was etherized and the biliary tract exposed. The gall bladder was removed and the common bile duct doubly ligated and sectioned. Observations were made repeatedly after obstruction of the biliary outflow during the period that the animals could be maintained in good condition, which varied from twenty-eight to thirty-eight days. Serum bilirubin was determined at various periods, using the van den Bergh technique.

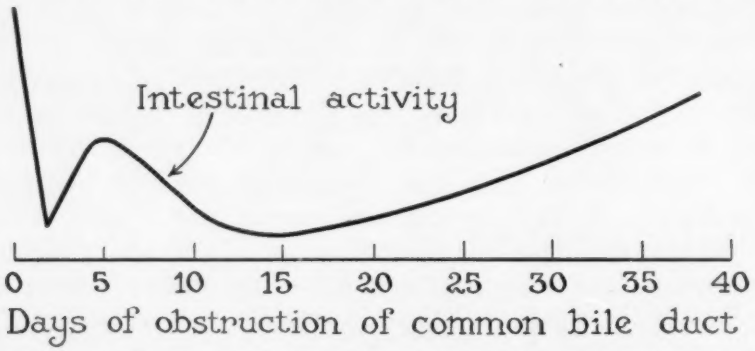

Fig. 1.—Diagrammatic representation of intestinal activity after ligation of common bile duct; curve made from kymograph records taken daily.

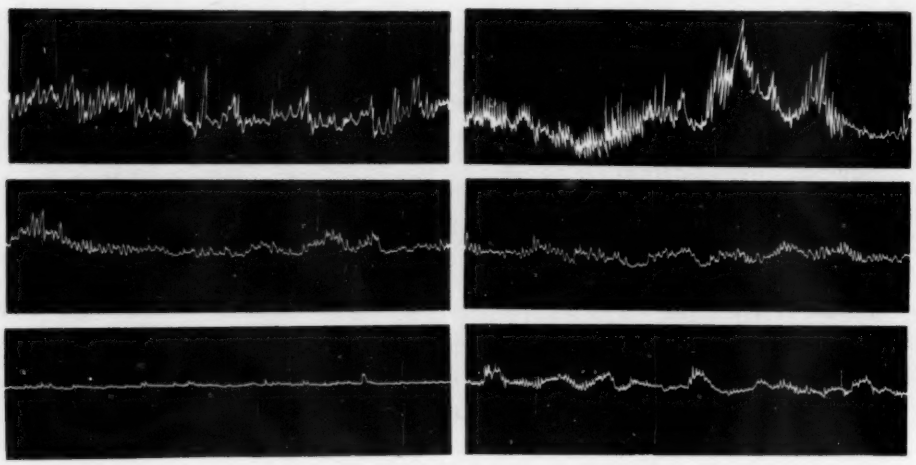

Fig. 2.—Intestinal activity of jejunal loop, 35 cm. from the ligament of Treitz. *A*, Control in fasting (left) and feeding (right) periods; *B*, five days after the obstruction of common bile duct; *C*, twelve days after the operation.

RESULTS

In general, the effect of obstruction of the biliary outflow on the motor response of the intestine was similar in all the animals, and the changes that occurred affected all the types of intestinal activity, such as regular and irregular segmentation, tonus waves, and peristalsis.

A marked decrease of activity occurred during the first twenty-four hours after operation, followed by quick recovery which reached a maximum on the fifth to sixth day of biliary obstruction (Fig. 1). Subsequently, intestinal activity progressively decreased and usually minimal activity was reached during the twelfth to fifteenth days after occlusion of the duct. The decrease of activity was more noticeable in the loops of jejunum than in the loops of ileum. The feeding reaction gradually diminished and sometimes did not occur (Figs. 2 and 3).

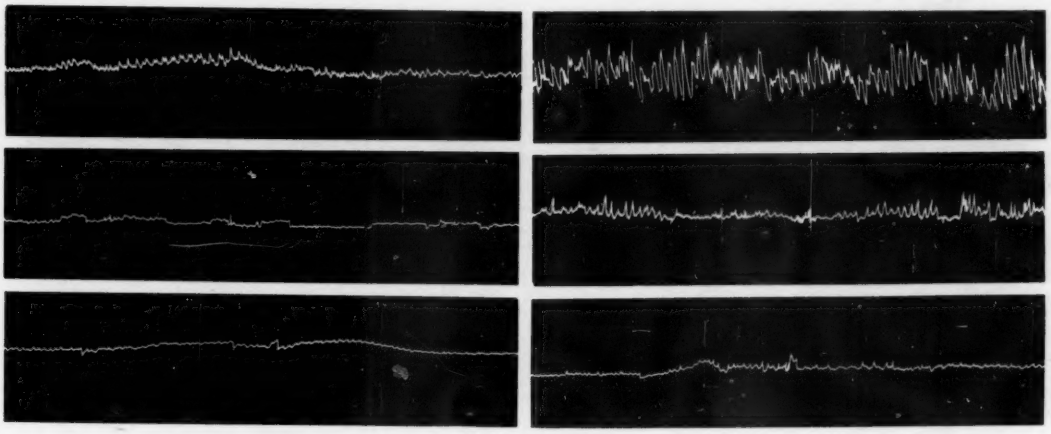

Fig. 3.—Intestinal activity of ileal loop, 15 cm. above the ileocecal valve. *A*, Control in fasting (left) and feeding (right) periods; *B*, six days after the obstruction of the common bile duct; *C*, fifteen days after the operation.

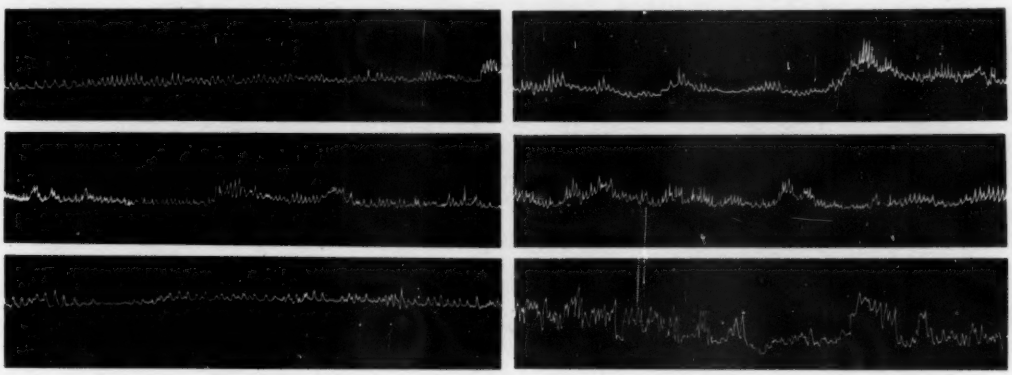

Fig. 4.—Same dog as in Fig. 2. *A*, In fasting (left) and feeding (right) periods, eighteen days after the obstruction of common bile duct; *B*, twenty-two days after the operation; *C*, thirty-eight days after the operation.

The periods of quiescence in the fasting period were often markedly prolonged. A definite increase of activity began between the fifteenth and twentieth days after operation, which was progressive throughout the remaining period during which observations were made, but never reached the amount that had occurred before ligation of the duct (Figs. 4 and 5). No changes of the frequency of the contractions were observed either before or after the obstruction of the duct.

The bilirubinemia increased gradually, reaching a maximum on the eighth or tenth day after operation. It subsequently progressively decreased to the end of the period of observation. There appeared to be an inverse relation between the changes of intestinal activity and the amount of bilirubinemia (Fig. 6).

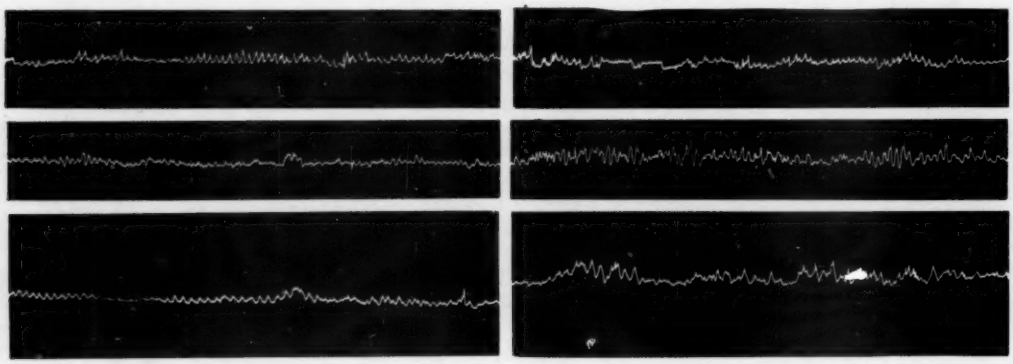

Fig. 5.—Same dog as in Fig. 3. *A*, In fasting (left) and feeding (right) periods, twenty days after the obstruction of the common bile duct; *B*, twenty-four days after the operation; *C*, thirty-five days after the operation.

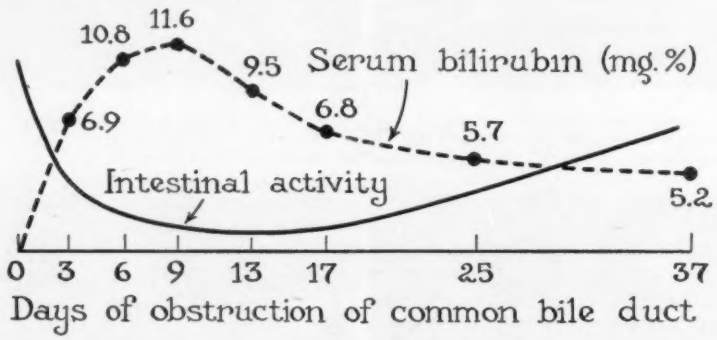

Fig. 6 (Dog 6).—Diagrammatic representation of the curves of bilirubinemia and intestinal activity after obstruction of common bile duct.

COMMENT

The sudden decrease of intestinal activity noted immediately after operation was apparently an effect of the anesthetic and operative procedures, and is in accord with previous observations. Golden found that intestinal activity is decreased greatly or abolished during surgical anesthesia but recovery often occurs with surprising rapidity. Mann and Wakim noted that an almost complete cessation of intestinal activity occurred for a few hours after ether anesthesia and exploratory operation involving exposure of the entire gastrointestinal tract for fifteen to twenty minutes. In each of the foregoing investigations, normal movements of the intestine were resumed within twenty-four hours after anesthesia. The changes of intestinal activity that were noted in this investigation after the animals had recovered from the immediate effects of the operation would appear to be the result of obstruction to the biliary outflow.

The changes of intestinal activity that were noted in the animals that had obstructive jaundice might be owing to the lack of bile in the intestinal tract, accumulation of the biliary constituents in the blood, or both. Gutzeit and Kuhlbaum expressed the belief, from the results of their study on obstruction of the common bile duct, that lack of bile in the intestine is the primary factor for the decrease of intestinal activity. Horrall and his co-workers, using fasted dogs that had both duodenal and gall bladder fistulas, and several balloons inserted into the gut, concluded that the flow of bile into the intestine is an important factor in maintaining intestinal activity. Haney, Roley, and Cole came to the same conclusion as a result of their study on the local action of bile in Thiry-Vella loops. On the other hand, Ackerman, Curl, and Crandall, working with dogs in which bile was excluded from the intestine by anastomosis of the gall bladder to the right renal pelvis and ligation of the common bile duct, observed by means of the opaque meal that the motility of the intestine was not affected by the lack of bile. In one of the animals in our series, a spontaneous external biliary fistula developed nine days after occlusion of the common bile duct. Intestinal activity increased to a considerable extent after the restoration of the flow of bile even though the bile was excluded from the gut.

There is some evidence that the retained biliary constituents, particularly the bile salts, in the blood may be responsible for the decreased intestinal activity in obstructive jaundice. Schroeder, using guinea pigs, and Chabrol, Lemaire, and Cottet, using dogs, observed that the intravenous injection of bile or bile salts produced a great decrease and sometimes cessation of intestinal activity. Berti and Bernucci mentioned that small doses of bile given intravenously to dogs produced a marked delay of passage of the opaque meal through the intestine while large doses caused an acceleration of the intestinal transit. We injected gall bladder bile of the dog (0.5 c.c. per kilogram) and pure bilirubin (30 mg. per kilogram) intravenously into normal dogs and noted a definite decrease of intestinal activity. While the effect of bile salts when administered intravenously in a single dose may not be the same as when they accumulate gradually in the blood stream after obstruction to the biliary outflow, the depressing action of these substances on intestinal activity is suggestive. The fact, previously mentioned, that the changes of intestinal activity after obstruction of the common bile duct appear to have a relation with the changes of amount of bilirubin retained in the blood also suggests that the retained biliary constituents of the blood may be of importance in causing the alteration of the motor mechanism of the intestine that we observed. In this connection it should be noted that Snell, Greene, and Rowntree found that the fluctuations of the amount of bile salts in obstructive jaundice were similar to the changes in bilirubinemia.

SUMMARY

Obstruction of the common bile duct was followed by a marked decrease of activity in exteriorized loops of jejunum and ileum during the first two weeks after operation. Subsequently, intestinal activity increased but never equaled the normal amount in the jaundiced animals. The decrease of intestinal activity occurred in both the fasting and digesting states but was more noticeable in the latter. It is suggested that the biliary constituents retained in the blood, particularly the bile salts, may be of importance in causing the observed alteration of intestinal activity.

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THE EFFECT OF INTRAPERITONEAL INJECTION OF GASTRIC, STRANGULATED INTESTINAL AND APPENDICAL LOOP CONTENT UPON THE LEUCOCYTE COUNT

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IF ANY one of a number of protein substances, of which peptone is an example, be injected intravenously into animals, there occurs within a few minutes a striking reduction in the number of leucocytes in the circulating blood, and this reduction in leucocytes is due mainly to the disappearance of the granular forms. After a period of several hours, the leucocytes increase and often reach a level much higher than normal.

Wyssocowitsch,[19] in 1886, was apparently the first one to note this phenomenon, and from that time until the present there have been many explanations as to the cause of the leucopenia.

Löwit,[10] in 1892, believed that there was an actual destruction of the cells because he found some degenerated white blood cells in the circulating blood, and he believed that various organs were able to break up the leucocytes. He injected peptone, nucleins, or sodium urate, and found the results to be the same. The leucocytosis which followed he considered to be a response from the bone marrow.

Goldscheider and Jacob[4] produced leucopenia by the injection of extracts of spleen, thymus, or bone marrow, and were of the opinion that the leucopenia was due to the collection of the white blood cells in the capillaries of the lungs, liver, and spleen as a result of a negative chemotactic influence on the leucocytes exerted by the injected substances. Schultz,[14] Gräff,[5] and Wells[18] held similar views.

Silverman[15] and Schilling[13] believed that the leucocytes collected in the capillaries as a result of mechanical obstruction of the capillaries, caused by swelling of the endothelial cells, and that the leucocytes were thus strained out of the circulation and kept in the organs.

Bruce[1] made sections of lungs, spleen, and liver during the period of leucopenia and found that there was a great increase in the number of leucocytes present in these organs.

Ewing[3] found that the normal liver contained 17,400 leucocytes per cubic millimeter, but during leucopenia the number rose to 40,000 leucocytes per cubic millimeter.

Doan and his co-workers[2] injected 1 Gm. of sodium nucleinate into rabbits and produced an immediate leucopenia which lasted several

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hours and which was then followed by a marked leucocytosis which persisted up to four days. Leucopenia was found in blood taken from the right heart, from the peripheral vessels, and the lungs. They found an accumulation of neutrophile polymorphonuclear leucocytes in the parenchyma of the spleen. If the spleen were removed no leucopenia occurred, but leucocytosis set in immediately after the injection.

Wells[17] injected dead bacteria into rabbits and found a greatly increased number of leucocytes in the spleen and liver. He examined blood from the lungs and the bone marrow during the leucopenia and found a normal number of leucocytes present. He believed that the leucopenia was not due to negative chemotaxis or to mechanical obstruction to the passage of the leucocytes through the organs, but that it was a manifestation of a positive chemotaxis toward the bacteria which collected in the liver and spleen.

Webb[16] studied the leucopenia which occurs in horse serum anaphylaxis in dogs. He found no aggregation of leucocytes in blood from the liver, intestines, kidney, spleen, bone marrow, portal vein, or peripheral vessels. Blood from the pulmonary vein, however, he found to contain fewer leucocytes than blood from the pulmonary artery and sections from the lung showed collections of leucocytes in the capillaries.

Hill[7] made leucocyte counts on blood from the liver, from a peripheral vein, from the spleen, and from a mesenteric vein in dogs during peptone leucopenia and found that there was a leucopenia present in all samples. He could find no evidence of accumulation of leucocytes in the liver or the spleen.

Hill and Stoner,[8] while investigating the toxicity of the contents of strangulated bowel, found that a profound leucopenia followed within five minutes after the intravenous injection of such material into dogs. They found that the leucopenia preceded the clinical symptoms of such injections by as much as thirty minutes, while in some instances, leucopenia was present with no clinical symptoms. Leucopenia also followed injection of the content of normal bowel, of bowel content in simple obstruction, and of the transudate collected from obstructed and strangulated intestine. Stoner and Hill believe that this leucopenia is a manifestation of some type of shock, and that the symptoms produced (hyperpnea, prostration, tremor, vomiting) by injection of intestinal contents are probably signs of the same phenomenon. Although they did not make detailed studies of the differential count in these experiments, it seemed that the leucopenia was not as definitely due to a disappearance of the granular forms as is the case in peptone leucopenia.

Instances in which splenic, liver or other tissue substances, peptones, nucleins or intestinal contents are suddenly introduced into the blood by natural process are rare, but the invasion of some of these into the peritoneal cavity does commonly occur and the possibility that leucopenias similar to those produced by intravenous injections might result from intraperitoneal injections has not been investigated. Osler,[12] in

1912, stated that after perforation of the intestine in typhoid fever, the leucocyte count might rise, remain stationary, or suddenly fall. Livingston and Squires[9] studied fifty-five cases of typhoid fever in 1925 and found that there was a decided fall in the leucocyte count in 18 per cent after perforation but they did not specify whether more than one count was made after the perforation occurred. Harris and Feldheym[6] recently reported leucopenia in patients following the release of an obstructed loop of an intestine. The reduction was usually noted on the first to fourth postoperative days and often was very pronounced. In one patient, for instance, the count was 14,400 before operation and 1,950 on the third postoperative day. In another case, the preoperative count was 11,750 and on the first, second, third, and fourth postoperative days it was less than 3,000. They attributed the leucopenia to a toxin which was released from the obstructed bowel and which produced a temporary bone marrow paresis, and in their opinion the leucopenia was not related to sepsis and did not mean a bad prognosis.

Moon[11] introduced ground muscle pulp, brain, liver, or kidney into the peritoneal cavity of dogs and observed a progressive circulatory deficiency which had all the characteristics of shock. He found that hemoconcentration was present but did not report leucocyte counts.

EXPERIMENTAL PROCEDURES AND RESULTS

Group I

Intraperitoneal Injection of Intestinal Contents

In order to obtain the material injected into the peritoneal cavity of the four dogs studied in this group, strangulation of the bowel was produced in other animals as follows: Ether was administered by intratracheal intubation after induction in an ether box. No preliminary medication was given. Intestinal strangulation was produced by ligation of intestine, mesentery, and vessels with one-half-inch twilled cotton tape under aseptic conditions. The incision was closed in three layers (peritoneum and muscle, subcutaneous tissue, skin) with No. 8 cotton thread. The loop was removed sixteen hours after strangulation to forestall rupture and loss of the contents. These contents, usually 100 c.c. of bloody, foul-smelling fluid, were removed from an intact strangulated gangrenous loop of jejunum about two feet in length.

After the injection of intestinal contents all four dogs had a more or less lowered leucocyte count within fifteen minutes. Immediately upon injection the dogs became very ill, trembled, whined, and vomited. When released one hour after injection they were weak, nauseated, and dejected and all but one died in about eight hours. Five dogs were then injected with a coarsely filtered saline suspension of contents of normal bowel. In each case the content of the whole small intestine, which was milked out immediately after the death of an animal killed with ether, was mixed with saline solution and filtered through gauze.

This material was then injected intraperitoneally. These dogs did not appear as ill as the former group and when released appeared normal. The leucocyte drop following injection in this group (Table I, *B*) was usually as definite at fifteen minutes as in the former group (Table I, *A*), but a significantly greater proportion recovered.

TABLE I

INTRAPERITONEAL INJECTION OF INTESTINAL CONTENTS

A. Contents of Strangulated Loop

DOG NO.	W.B.C. NORMAL	AMT. INJECTED (C.C.)	W.B.C. 5 MIN.	W.B.C. 15 MIN.	W.B.C. 30 MIN.	W.B.C. 1 HR.	COMMENT
581	12400	20	12700	11000	3600	7500	Recovered
584	18100	100	18600	14950	10750		Died in 15 hours
574	15150	50	14200	11650	7900	6500	Became very ill upon injection
576	16800	50	12800	9650	7800	5150	Died during night

B. Contents of Normal Dog's Intestine

DOG NO.	W.B.C. NORMAL	AMT. INJECTED (C.C.)	W.B.C. 5 MIN.	W.B.C. 15 MIN.	W.B.C. 30 MIN.	W.B.C. 1 HR.	W.B.C. 1½ HR.	W.B.C. 24 HR.
575	17100	15	15400	16000				Recovered
578	8700	20	7850	6800				Recovered
577	31500	20	26500	18500	14600			26050
597	7750	50	5350	6150	6650	3250	1300	Died
598	8650	50	6650	4550	2950	1600	1000	Died

TABLE II

INTRAPERITONEAL INJECTION OF GASTRIC CONTENTS

DOGS NO.	605	606	594	715	716	718	717	719
W.B.C. NORMAL	8450	15000	13000	11050	10800	16300	11250	12450
Gastric Contents Injected								
15 min.	8150	13100	11150	10300	10300	14300	3150	10700
30 min.	6500	8600	11350	11900	11450	11800	3000	11300
60 min.	6000	12700	5700	8250	13950	12500	3100	10600
1½ hr.	6700	12700	3850	6850	11150	14700	3900	9450
2 hr.	6550	11650	5750	7800	10950	11950	2700	8400
2½ hr.		11400	2450	6800	11200	16500	4100	6400
3 hr.	7850		7400					4400
4 hr.			11700					7400
5 hr.			14750					7900
21 hr.			20150	21550	29200	45300	9400 Died in 49 hr.	19400

Remarks

Dog No. 605, Dog No. 594: Given 20 c.c. unfiltered gastric contents removed from healthy dog after meal of white bread

Dog No. 606: Given 20 c.c. filtered saline suspension of contents of stomach removed from dog under ether anesthesia

Dog No. 715, Dog No. 716: Given 50 c.c. of unfiltered gastric contents removed one-half hour after histamine stimulation; stomach was here lavaged with 300 c.c. of distilled water before injection of histamine and removal of contents

Dog No. 718: Given 25 c.c. of filtered gastric content; stomach was not lavaged (otherwise same as Dogs No. 715 and 716)

Dog No. 717: Given 50 c.c. of unfiltered contents from unlavaged stomach removed one-half hour after histamine stimulation; this dog, a female Scottie, in estrus, died 49 hours after the injection

Comment and Conclusions From Table I.—When the contents of strangulated bowel are injected intraperitoneally into another animal there is a marked systemic reaction which usually results in death. About thirty minutes after the injection there is a pronounced drop in the leucocyte count in the peripheral blood.

When the contents of normal intestine are injected in the same manner, the systemic reaction is less severe and the animal usually recovers. The fall in leucocytes is not constant or pronounced but usually occurs.

Group II

Intraperitoneal Injection of Gastric Contents

In order to determine if leucopenia might occur following rupture of a viscus, we procured gastric contents by lavage of a dog's stomach one-half hour after eating white bread (Dogs No. 605 and 594) and by washing a stomach (removed from dog under ether anesthetic for one hour) with saline solution (Dog No. 606).

White bread was used as a stimulating meal to avoid the side effects which would probably follow intraperitoneal injection of the meat extract test meal usually used. However, because white bread is such a poor secretagogue, it was decided that histamine diphosphate (2.08 mg. intramuscularly) should be used in the remainder of the experiments in Group II. Gastric contents were removed one-half hour after injection of histamine and injected into Dogs 715, 716, 717, 718, and 719 after treatment as indicated in "Remarks." Results of such injections can be seen in Table II. All dogs in this group lived with the exception noted in Table II.

Comment and Conclusions From Table II.—When gastric contents are injected intraperitoneally a drop in leucocyte count occurs in over one-half the animals. This drop is not constant in degree or time of occurrence. The systemic reactions in this group were not as severe as in Group I. All animals in this group lived.

Group III

Rupture of the Appendix

The appendices of five dogs were ligated in such a way that venous return was stopped. This operation was done aseptically with intratracheal ether anesthesia after induction in an ether box, and with no previous medication. A cutting suture was fixed to the appendix in the distal one-third. At six hours the appendix was swollen to three times its former size and was black and gangrenous. *At this time these dogs had a leucocytosis.* Rupture of the appendix (produced by pulling the cutting suture) resulted in a drop in white blood count in three out of five animals, and an increase in signs of illness.

Comment and Conclusions From Table III.—When a gangrenous appendix is made to rupture by the use of a cutting suture the leucocyte count often falls. It is only rarely that it goes higher than it was

TABLE III

WHITE BLOOD COUNT FOLLOWING RUPTURE OF APPENDIX

DOG NO.	601	602	604	607	608
NORMAL	9450	9100			13500
Appendix freed from mesentery, ligated, and cutting suture affixed					
6 hr. Postoperative	18700	15650	18400	19000	29900
Cutting suture removed, appendix ruptured					
5 min.	16350	16250			
15 min.	16100	14250			
30 min.		15150	21200	10550	29050
60 min.	14800	13350	18350	6950	26000
1½ hr.		8350	15950	1850	26050
2 hr.		6600	19400	900	18000
2½ hr.		7500	16400	1900	17500
3 hr.		4650	21400	1950	18300
3½ hr.		3950	18900	3250	17500
4 hr.		4000	17500	3650	16650
4½ hr.		1400		2200	
5 hr.		2050	20560	Died	19150
5½ hr.					24650
6 hr.			17150		23650
6½ hr.			21850		22400
Died at	22 hr.	20 hr.	16 hr.	5 hr.	24 hr.

before rupture. The drop in the leucocyte count is not constant in degree or time of occurrence but occasionally a marked leucopenia develops.

It might be expected that the blood picture following rupture of a gangrenous strangulated appendix would be similar to that produced by injection of contents of a strangulated jejunal loop. However, symptoms appeared more rapidly in the latter, the leucopenia appeared more quickly, and the length of life was much shorter.

Dogs in the first (Table I, *A*) and the last (Table III) of these experiments seemed prostrate. They shivered, vomited, and their extremities became cold. They were acutely ill.

SUMMARY

In an attempt to simulate the conditions which exist after an acute abdominal catastrophe such as the rupture of a peptic ulcer, the rupture of an acutely inflamed appendix, or the rupture of a section of gangrenous bowel, the above series of experiments were performed. In addition to the severe systemic reaction produced on the animals by these procedures, there was a notable tendency for the leucocyte count in the peripheral blood to fall. It seems reasonable to assume that such a drop in the leucocyte count may occur under similar circumstances in man, and that it has not been previously detected except in isolated cases is probably due to the fact that blood studies were not made at the appropriate times. If such a fall in the leucocytes does occur in man it may explain some of the unexpectedly low leucocyte counts which are not infrequently found.

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TREATMENT OF SMALL BOWEL OBSTRUCTION*

Procedure Used at the University of Minnesota Hospitals

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ALTHOUGH improved techniques in the management of obstruction of the small intestine have cut the mortality to a quarter of that obtaining twenty years ago, this affliction still commands a mortality rate between 18 and 20 per cent in the leading surgical clinics (Table I).

Discussion of the improvement which has occurred generally requires some discussion of the factors involved in small bowel obstruction. Anatomically speaking, there are but two types of intestinal obstruction, the simple obstruction (that which bars only the passage of intestinal content), and the strangulating obstruction (that which prevents also the flow of blood to or from the involved segment). Obstructions of either type may be partial or complete, according to the degree of interference with flow or intestinal content.

MECHANISMS OF OBSTRUCTION

The mechanisms of obstruction, in the order of frequency in which they are seen at this clinic, are as follows: (1) External hernia; (2) intussusception; (3) adhesive bands, frequently found in association with (4) adhesions of the bowel to other structures with the formation of kinks which render the obstruction ever more complete as the degree of distention increases; (5) foreign body or obturation obstruction, in the majority of cases due to large gallstones reaching the gut by cholecystenteric fistulae (Rigler, Borman, and Noble); (6) volvulus; (7) internal hernias; (8) mesenteric vascular occlusion, and (9) neoplasms. Hernias, adhesive bands, volvulus, and, of course, mesenteric vascular occlusion are prone to lead to gangrene of the involved bowel and fatal peritonitis if operative intervention is not prompt. Intussusception also leads frequently to gangrene of the intussusceptum, but the ensheathing intussuscipiens remains viable and delays or prevents infection of the peritoneal space.

It is the obstructions due to adhesions of the intestine to other structures in the abdomen which are successfully treated by conservative management alone.

THE MANNER OF ONSET OF SYMPTOMS AND SIGNS

Pain and Cramps.—The onset of obstruction is followed within a short time by accumulation of fluid, food, and gas above the site of

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closure. The mechanism of pain production is twofold, either a rapid stretching of the intestinal musculature or resistance to the strong peristaltic contractions of that layer. In many instances, particularly in partial simple obstructions, this accumulation is gradual, and the inevitable stretching of the bowel wall is sufficiently slow so that pain is not appreciated by the patient except at the moments when peristaltic rushes in the gut reach the distended area. This pain, occurring with the arrival of peristaltic rushes at the distended bowel, is the cramp commonly associated with intestinal obstruction.

The frequency of rhythmic pendular contractions in the jejunal muscle has been shown by Alvarez and associates to be about twice that of muscle from the terminal ileum. There is some reason to believe that the frequency of periods of increased local muscular activity, corresponding to peristaltic rushes, also follows a gradient. In cases in which the level of intestinal obstruction is in doubt, the frequency of cramps may occasionally be of aid, the cases presenting intervals less than 3 to 4 minutes tending to have high obstructions and those of eight to ten minutes, low ones. The appreciation of pain between cramps is probably a function of the rapidity with which material is accumulating above the obstructed area.

Borborygmus.—Gas is normally present throughout the intestinal canal, but in the small intestine, where activity is great, it usually does not collect in pockets large enough for x-ray visualization (Wangensteen). With the onset of obstruction and stagnation above, gas in quantity collects above a small quantity of fluid. Each peristaltic rush forces a little more gas and fluid into this closed space, resulting at first in gurgles, audible with the stethoscope at the moment of the height of the cramp. As the volume enclosed within the bowel above the obstruction increases, the wall of this bowel is stretched increasingly more tightly, and the pitch of the audible sound coming with each cramp rises correspondingly until it becomes the familiar tinkle or borborygmus of established intestinal obstruction.

Vomiting.—Distention of the small bowel is accompanied by reverse peristalsis from time to time, and regurgitation into the stomach with vomiting occurs. In complete ileal obstructions of two to three days' duration, the vomitus begins to assume a fecal character; the fecal vomiting appears later in partial obstructions, if it appears at all.

X-ray Signs.—Although some of the distention is due to fluid in the gut, the greater part of it comes from swallowed air, as Wangensteen and Rea have shown in dogs. The presence of this gas within the bowel is not helpful in producing physical signs of obstruction, but because of it x-ray diagnosis is accurate in the majority of cases. Scout films of the abdomen reveal a ladderlike arrangement of gas-filled coils of the small intestine. In obstructions of several days' duration, the ratio of fluid to gas increases, and upright films of the abdomen show definite

fluid levels in the distended loops of bowel. Furthermore, the degree of absence of gas visible in the colon is a good index of the completeness of obstruction, provided gas has not been injected with enemas. Although a barium meal outlines the obstructed bowel very beautifully, the barium sulfate is apt to cake and increase the completeness of obstruction. It is therefore to be condemned.

Shock.—In the case of unrelieved strangulating obstructions, gangrene of the bowel wall occurs, and peritonitis develops. Inasmuch as venous compression is greater than arterial, large enough quantities of blood may be lost into the lumen of the bowel and into the peritoneal space to cause early death from shock (Wangensteen).

Back Pain.—In addition to the symptoms already outlined as characteristic of simple small bowel obstruction, patients with strangulating obstructions are apt to experience sudden onset of the attack with severe, frequently constant pain, often low in the back as well as in the abdomen. In our experience, the low back pain can be ascribed to tension on the mesentery resulting from passage of gut into herniae, around adhesive bands, etc.

Peritoneal Irritation.—The escape of bloody fluid or seepage from the gangrenous gut leads to marked tenderness in the abdomen, with muscle spasm and rebound tenderness. Elevation of temperature and leucocytic count follow in a few hours.

Palpable Loops.—In certain cases the degree of obstruction is slight enough so that compensatory hypertrophy of the intestinal muscle above is adequate to prevent decompensation. Between these extremes are many patients with partial obstruction complete enough to give the symptoms already mentioned, but in whom sufficient hypertrophy occurs to permit visualization of the outlines of loops on the abdominal wall and palpation of them with the examining hand. This degree of hypertrophy can develop in seven to ten days of chronic partial obstruction.

CAUSES OF DEATH IN INTESTINAL OBSTRUCTION

The cause of death in bowel obstruction has been extensively studied and is not settled. In high obstructions, sufficient water, acid, and chloride are lost to lead to death in short order from dehydration, hypochloremia, alkalosis, and uremia (Hartwell and Hoguet). With obstructions at progressively lower levels, these changes become less evident, and in terminal ileal obstruction they are almost inconsequential. In these cases the deaths are due to one of two causes, either (1) prolonged distention or (2) gangrene of the bowel wall, the presence or absence of gangrene depending upon whether a simple or a strangulating obstruction is present.

The mechanism by which distention proves lethal is hotly debated. Sperling showed that a sustained intraluminal pressure of 15 cm. of water is capable of injuring the bowel wall through vascular stasis,

thus altering the histology, impairing the viability of the gut, and opening a new avenue of absorption, namely, transperitoneal migration. That products of bacterial action may be the noxious substances so absorbed is indicated by the report by Poth and Knotts that the placing of succinyl sulfathiazole in closed loops prolongs survival of the animal 500 per cent. Afferent nervous impulses have been considered lethal (Antoncic and Lawson) but this has not been corroborated as yet, although nausea and vomiting with secondary disturbances of chemical balance may be so produced (Burget and co-workers).

In the case of strangulating obstructions, death may be due to shock or to peritonitis.

SUMMARY OF ESSENTIAL POINTS IN DIAGNOSIS OF SMALL BOWEL OBSTRUCTION

The essentials of diagnosis of simple small bowel obstruction, therefore, are:

1. Crampy abdominal pain
2. Vomiting, fecal if late
3. More or less abdominal distention
4. Minimal abdominal tenderness, localized
5. Borborygmi with the peaks of the cramps, and
6. X-ray findings on flat plate of the abdomen

Strangulating obstruction presents:

1. Pain, usually crampy, often persisting between cramps (often in the back)
2. Vomiting, fecal if late
3. Distention
4. Tenderness, usually over a considerable area
5. Borborygmi, unless the case is late enough for paresis of the gut to have occurred
6. X-ray findings
7. Spasm and rebound tenderness, and
8. Shock (occasionally).

TREATMENT OF SMALL BOWEL OBSTRUCTION

The preceding discussion indicates that proper therapy should consist of (1) restoration of the patient's water and mineral balance and of his blood pressure to normal, (2) relief of distention (either by intubation of the small bowel or by removal of the obstructing mechanism), and (3) resection of such gangrenous bowel as may be present. At clinics such as the Massachusetts General Hospital, where two-thirds of the patients are admitted in the first forty-eight hours of the attack, the emphasis has been laid on early operation before the onset of distention (McKittrick).

Intubation.—At this hospital, on the other hand, enough of the patients have arrived later than forty-eight hours after the onset of symptoms to have drawn attention to alleviation of already present distention as the primary problem. Wangensteen and Paine developed the

siphon suction apparatus now in general use in this country, and have shown that constant suction on an inlying duodenal tube will decompress cases of simple small bowel obstruction. More recently, Miller and Abbott have perfected a twelve-foot double lumen tube tipped with a balloon which can be inflated through one lumen to facilitate passage of the tube down the small intestine..

Johnston, Abbott, and others have outlined many maneuvers which can be used to pass the tip of the Miller-Abbott tube into the duodenum. Abbott has recently reported the use of a stylet of leader wire to stiffen the tube sufficiently to cause it to pass into the pylorus directly, the stomach being dilated with 500 to 600 c.c. of air. This procedure has worked nicely in five of the seven cases in which we have tried it.

Metabolism.—Hartwell and Hoguet, in 1912, called attention to the value of large amounts of normal salt solution in the treatment of high obstruction, and clinical applications were made by Haden and Orr, and others. With this impetus, efforts have been made to give an adequate caloric intake by the paraoral route, as outlined by Coller and Maddock. At this hospital, a caloric intake in excess of 1200 calories a day is accomplished by the administration of 20 per cent dextrose solution not faster than 150 c.c. per hour. A proper level of plasma electrolytes and blood urea nitrogen is usually quickly attained by giving sufficient salt and water to permit excretion of a liter of urine and 2 to 5 Gm. of salt daily. The available vitamins are given parenterally. In patients unable to take food by mouth over a considerable period, the use of amino acid preparations and small amounts of human plasma, such as 100 c.c. daily, have become routine in an effort to maintain a normal nitrogen balance. Larger quantities are necessarily given if the plasma protein level is found to be low.

Operative Procedures.—Most important, the patient is watched closely at four- to six-hour intervals for evidences of peritoneal irritation or failure of decompression. Scout films of the abdomen are made from time to time. If a tube cannot be passed into the small bowel in forty-eight hours, and the obstruction persists with continuing distention, catheter enterostomy without exploration is the usual procedure. If passage of the tube is not followed by satisfactory decompression for any reason, catheter enterostomy is also indicated. In patients in whom satisfactory decompression is not followed by relenting of the obstruction, exploration as an elective procedure is undertaken to restore intestinal continuity.

If signs of peritoneal irritation appear, exploratory laparotomy is in order, regardless of the degree of distention. In this type of patient, sufficient hemorrhage is likely to have occurred to precipitate shock, and blood or plasma in quantity should be at hand before undertaking surgical intervention.

In patients in whom a Miller-Abbott tube has been passed into the lower reaches of the small bowel, feeding by mouth of a liquid or low residue diet is often feasible, even if the obstruction fails to relent. Such procedure greatly facilitates the metabolic preparation of the patient for operation.

Signs of Relenting of the Obstruction.—The appearance of gas in the colon, as recognized by x-ray scout film, or the passage of gas by rectum, indicates that the obstructive mechanism has relented. It is customary here to allow the tube to remain in place an additional day or two and to check the tolerance of the patient to cessation of suction for one-half day before withdrawal of the tube.

Oxygen.—Fine and associates have demonstrated that respiration of high concentrations of oxygen aids in diminishing the gaseous distention in cases of intestinal obstruction. The method has proved of aid in some cases here, but suction is a much more direct approach to the problem of distention, and oxygen is used only occasionally as an additional measure in those cases in which suction alone has proved not quite adequate.

RESULTS

In the period from June 1, 1938, to April 1, 1942, 130 patients suffering from obstruction between the ligament of Treitz and the cecum were treated at the University of Minnesota Hospitals. The present detailed study is concerned with 110 of these patients, most of the remainder being patients in whom obstruction of the small intestine was

TABLE I

MORTALITY RATES AS THE RESULT OF OBSTRUCTION OF THE SMALL INTESTINE

Lewis (Cook County)	21.7%	
Schlicke (Mayo Clinic)	17.8%	
McKittrick and Sarris (Mass. Gen. Hospital)	20.0%	
Johnston (Wayne and Detroit Receiving)	9.5%	(excludes strangulation obstruction)
Leigh (College of Physicians and Surgeons, New York)	13.2%	(selected cases)
Wangensteen, 1939 (Univ. of Minn. Hospital)	17.9%	

TABLE II

CASES EXCLUDED FROM THE PRESENT STUDY BECAUSE OBSTRUCTION WAS ONLY AN INCIDENT IN DEATH FROM OTHER CAUSE

CAUSE OF DEATH	NUMBER OF CASES
Carcinomatosis	7
Congenital anomalies in newborn infants	5
Abdominal abscesses perforating the bowel	2
Subphrenic abscess and sepsis from ruptured appendix	1
Severe sepsis from ruptured appendix*	1
Tuberculous peritonitis	1
Riding embolus at bifurcation of aorta followed terminally by mesenteric thrombosis	1
Total	18

*This patient survived the obstruction, but was still seriously ill from other complications of appendicitis at the time of this report.

a minor incident in death from some other cause. These special causes of death are given in Table II.

Inasmuch as the surgical treatment of obstruction is under consideration, two patients who died in the hospital early in the study period without having been seen by a surgical consultant are also excluded. One of these had a gallstone obstruction, the other an intussusception.

The corrected figures are shown in Table III. These are limited to small bowel obstruction cases treated by the surgical service in the three years and ten months under consideration. The variance between the case mortalities indicates that a smaller percentage of repeated admissions has occurred for obstruction recurring after conservative therapy, the result of a somewhat more aggressive attitude in the general treatment of intestinal obstruction.

TABLE III

SMALL BOWEL OBSTRUCTION

(UNIVERSITY OF MINNESOTA HOSPITALS)

JUNE 1, 1938, TO APRIL 1, 1942	
Total cases	117
Total patients	110
Total deaths	17
Case mortality	14.2%
Patient mortality	15.5%
JUNE 1, 1931, TO JUNE 1, 1938	
Case mortality	14.7%
Patient mortality	17.9%

The cases have been broken down into groups for study. Inasmuch as the use of the term strangulation implies constriction of blood supply and not yet loss of viability, it is somewhat ambiguous from the point of view of prognosis. We, therefore, have divided the cases into those which proved to have gangrenous bowel present at the time of admission or later, and those which did not have gangrenous bowel present. A second division separates those treated entirely by conservative methods from those treated by any type of operative procedure.

TABLE IV

SIMPLE SMALL BOWEL OBSTRUCTION

CONSERVATIVE THERAPY

INTUBATIONS OF THE SMALL BOWEL*				
TYPE OF TUBE	INTUBATION NOT ATTEMPTED	SUCCESSFUL INTUBATION	FAILURE IN INTUBATION	FAILURE FOLLOWED BY ENTEROSTOMY
Wangensteen	2	13	5	9
Miller-Abbott	0	11	3	7
Total cases			32	
Total patients			30	
Deaths			0	

*The figures do not check because of two patients in whom failure occurred with the Wangensteen tube, and in whom success was achieved with the Miller-Abbott tube.

Conservative Treatment of Simple Obstructions.—Table IV shows the cases treated only by conservative therapy in which no gangrenous bowel was present at any time. It is evident that this is a small-risk form of treatment if one can be reasonably assured that the obstruction is of a type which will relent. Some patients, ten in all, obtained relief and recovery with gastric suction alone. When the Wangensteen tube was used, success in intubation of the duodenum was achieved in thirteen out of twenty-nine attempts. Intubation was accomplished in a somewhat greater proportion of trials with the Miller-Abbott tube. The Miller-Abbott tube is now used routinely on all cases of obstruction of the small intestine treated at this clinic.

Operative Treatment of Simple Obstructions.—The second major group of patients consists of cases treated operatively who proved to have no gangrenous bowel present. Table V presents the data for this group.

TABLE V

SIMPLE SMALL BOWEL OBSTRUCTION
OPERATIVE THERAPY

Total cases	62
Total patients	57
Total deaths	8
Case mortality	12.9%
Patient mortality	14.0%

Among the operations performed, catheter enterostomy first draws attention, for it is the procedure adopted when conservative management fails. The procedure is done by an almost perfectly aseptic technique, placing the catheter in an emptied segment of gut after the Witzel

TABLE VI

SIMPLE SMALL BOWEL OBSTRUCTION
CATHETER ENTEROSTOMY*

Indications	
Failure to pass Wagensteen tube	6
Failure to pass Miller-Abbott tube	4
Failure to pass either tube	3
Fear of strangulation	1
Failure of Wangensteen tube to decompress	1
Blind loop	2
Failure of a previous enterostomy	1
Function	
Good	15
Poor	2
Patient died too soon to estimate	1
Mortality	
From enterostomy itself	2†
From other causes after successful enterostomy	4
Total cases	18
Total patients	17

*The total number of enterostomies performed is not indicated in this table, as some have been done prophylactically at the time of resection of gangrenous bowel, etc.

†One patient died of perforation of the bowel at operation. The other developed a high intestinal fistula and died of anasarca thirty-eight days after operation.

TABLE VII

SIMPLE SMALL BOWEL OBSTRUCTION

OPERATIVE PROCEDURES	CASES	DEATHS
Enterostomy alone	13	3[1]
Hernioplasty	19	1[2]
Intussusception, reduction of	11	0
Enterolysis	4	1[3]
Enteroanastomosis at an interval after enterostomy	2	1[4]
Resections	2	1[5]
Freeing of bands	2	0
Removal of gallstone alone	3	0
Enterolysis and resection	1	0
Enterostomy plus		
Lysis	1	1[6]
Removal of gallstone	1	0
Enteroanastomosis	1	0
Freeing of bands	2[7]	0

[1]Pneumonia at 12 days; rupture of the gut at operation; fecal fistula at enterostomy site with death from anasarca thirty-eight days postoperatively.
[2]Atelectasis five days postoperatively.
[3]Hemorrhage five days postoperatively.
[4]Anastomosis broke down (open anastomosis).
[5]Adhesive peritonitis preoperatively. Bowel healed well, but new adhesions formed. Patient died of lung abscesses, peritonitis, continued obstruction, and sepsis. Previous enterostomy.
[6]Pneumonia nine days postoperatively.
[7]In one of these cases, the distended bowel was decompressed at operation by Dennis and Wangensteen by an aseptic procedure suggested by them; it is being reported elsewhere (Wangensteen, 1942).

TABLE VIII

SMALL BOWEL OBSTRUCTION AND GANGRENE OF BOWEL
CONSERVATIVE THERAPY

Survival
Perforation of appendical abscess into bowel
Deaths
1. Postoperative strangulation around an appendicostomy performed for decompression in cancer of colon; obstruction not recognized
2. Postoperative strangulation of gut around old adhesive band. (Operation, amputation for diabetic gangrene.) Obstruction not recognized
3. Mesenteric venous thrombosis, admitted after six days
4. Intussusception, admitted moribund

Total cases	5
Deaths	4

pattern (Wangensteen). Such a catheter may be removed by simple withdrawal after it has served the purpose of decompression, and obstruction never occurs at the point of such a procedure. Table VI shows our experience with catheter enterostomy.

The various operative procedures performed on the patients with simple small bowel obstruction are indicated in Table VII.

Conservative Treatment of Patients With Gangrenous Bowel.—The third major group of cases with which we are concerned encompasses those treated conservatively in whom gangrenous bowel was present. The data are presented in Table VIII. The four deaths are not to be construed as arguments against conservative therapy, for in two the presence of obstruction was not recognized at all, and the other two patients were moribund on admission.

Operative Treatment for Patients With Gangrenous Bowel or Bowel of Doubtful Viability.—The final cases for presentation are those with gangrenous bowel or bowel of doubtful viability which were treated operatively. These are presented in Table IX.

TABLE IX

SMALL BOWEL OBSTRUCTION AND GANGRENE OF BOWEL
OPERATIVE TREATMENT*

Recoveries		
Resection of gangrenous bowel and primary anastomosis		10
Exteriorization with delayed closure		2
Inversion of small gangrenous area during hernioplasty		1
Deaths		
Reduction of gangrenous intussusception		2
Excision or exteriorization of gangrenous bowel in presence of gross pus or peritonitis		3
Total cases	18	
Total patients	18	
Deaths	5	
Mortality	27.8%	

*Among the cases recovering are included three infants with gangrenous intussusceptions, two of which were resected with primary anastomoses, and one of which was exteriorized, secondary closure being accomplished at a later date.

RESULTS IN THE RECENT PAST

In the past two years, it appears to us that the management of obstruction of the small intestine has improved materially at the University Hospitals, due to a series of influences. Improvements in the maintenance of metabolic balance by parenteral routes of administration seem to have played a part, and concentration of responsibility for the care of most of these patients in the hands of two persons (J. R. Paine and Dennis) has led to a more aggressive attitude with regard to surgical intervention. In the care of cases presenting gangrenous bowel, in particular, this policy has been fruitful, no death resulting in the last thirteen such cases. The use of the closed, aseptic anastomosis described by Dennis three years ago, and also of the local implantation of sulfonamides, recommended by Varco and collaborators, has played a role in this improvement, and results will be detailed elsewhere (Dennis, 1942).

If the forty-six-month period under study is broken roughly into two divisions, considering the past two years separately from the preceding twenty-two months, the data of Table X are obtained.

TABLE X

MORTALITY RATES FOR THREE CONSECUTIVE PERIODS
(AT THE UNIVERSITY OF MINNESOTA HOSPITALS)

	1931 TO 1938 (WANGENSTEEN ET AL.)	JUNE 1, 1938, TO APRIL 1, 1940	APRIL 1, 1940, TO APRIL 1, 1942
Number of cases	190	60	57
Number of patients	156	57	53
Number of deaths	28	11	6
Mortality:			
Case (%)	14.7	18	10.53
Patient (%)	17.9	19.3	11.32

The six deaths which have occurred in the past two years are worthy of note. Three patients died because of faulty surgery, such as accidental rupture of the bowel during an enterostomy, poor placement of the catheter, and leaving defects in the abdomen through which internal herniations subsequently occurred. Two died from failure to recognize the presence of strangulating obstructions as postoperative complications, one after ileosigmoidostomy with appendicostomy for multiple carcinomas of the colon, and the other after amputation for diabetic gangrene. One died of adhesive peritonitis, despite repeated decompressions, following decompression of an obstructed colon by colostomy.

In the last year of the study period, twenty-seven patients have been treated with loss of but two, a mortality rate of 7.4 per cent. In this period there were five resections for gangrenous bowel. One of the deaths was due to failure to recognize obstruction at all and the other to adhesive peritonitis.

RELATION OF AGE AND DURATION OF ILLNESS TO OUTCOME

McKittrick is inclined to stress the importance of early treatment, and had no deaths in forty-three cases operated upon within twenty-four hours of the onset of the disease. Forty-four per cent of our cases arrived at the hospital in the first twenty-four hours of the illness; in this group the mortality was 7.8 per cent. Seventeen per cent arrived in the second twenty-four hours and presented a mortality of 10 per cent. About 39 per cent arrived after forty-eight hours; in this group the mortality was 20 per cent. Early treatment, therefore, is of great importance whether the conservative or the operative approach is employed.

McKittrick found that advanced age is a great hazard to the patient with small bowel obstruction. Our experience over the period under study shows a mortality rate of 13.2 per cent for those under 60 years of age, a rate of 30 per cent for those from 60 to 70 years old, and a rate of only 7.1 per cent for those over 70 years old. These figures imply that our series is too small for conclusions.

COMMENT

It at first appears that less emphasis is being placed upon conservative therapy at this clinic than was the case at the time of the last report (Wangensteen, Rea, Smith, and Schwyzer). The reason for this is largely the success which has attended anastomosis in the presence of marked degrees of obstruction and gangrenous bowel, when performed by the closed technique. Inasmuch as the intestinal bacterial count can be reduced by oral administration of succinyl sulfathiazole preoperatively in operations *of election* (Poth), the closed anastomosis with local sulfathiazole implantation finds its field of greatest usefulness in *emergency* obstruction cases.

Success in dealing with cases with frank gangrene has encouraged us to be more aggressive in operating upon cases presenting only minimal evidences of peritoneal irritation. The excellent record of McKittrick and Sarris in operating upon early cases of small bowel obstruction as emergencies is also a strong factor favoring this procedure. We feel that it is only by an intelligent, combined use both of the improved methods of small intestinal intubation and of the newer surgical methods of coping with the problem that further reductions in mortality can be accomplished.

CONCLUSIONS

1. Despite improvement in the past two decades, small bowel obstruction still commands too high a mortality rate.
2. Treatment consists of:
   a) proper water and salt administration
   b) decompression of the distended gut by nasal tube, or by catheter enterostomy, if the tube is inadequate
   c) parenteral administration of sugar, plasma, etc., to give a complete metabolic balance
   d) resection of gangrenous bowel, if present, and
   e) late operative restoration of continuity of the bowel if the obstruction fails to relent spontaneously
3. Results of treatment of 110 patients with small bowel obstruction during the period from June 1, 1938, to April 1, 1942, are presented.
4. Our treatment of this condition is apparently improving.

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PENETRATING GUNSHOT AND STAB WOUNDS OF THE ABDOMEN

A Review of 336 Cases

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AN ANALYSIS of sixty-nine gunshot and stab wounds at the Louisville City Hospital was made by McKeithen[1] in 1927. The present series covers the past ten and one-half years. One of us (J. E. H.) has personally supervised this branch of traumatic surgery since late 1938. During the period from January, 1930, to July, 1941, 227 patients with gunshot and 184 with stab wounds of the abdomen have been admitted to the hospital. Thirty-seven gunshot and thirty-eight stab wound cases have been discarded, leaving for the present study, 190 perforating gunshot and 146 perforating stab wounds of the abdomen.

The discards were made for the following reasons: Sixteen bullet and thirty stab wounds were found nonpenetrating upon exploration of the abdomen. All the patients with negative explorations recovered uneventfully. Twelve patients with bullet wounds and five with knife wounds expired upon reaching the ward or within a few minutes thereafter. It seemed reasonable to discard as moribund these patients dying in less than an hour of admission, since they were doomed regardless of treatment. It is to be emphasized, however, that nine other cases (eight gunshot and one stab), although never coming to operation, were included in the present series. All but one of these patients were admitted in the earlier years and all were vainly "treated for shock" for from one to six or eight hours until they finally died. Although not operative deaths, these cases were nonetheless surgical casualties, for no operative measures were taken to control the bleeding that was undoubtedly responsible for their collapse and death.

A third group of discards consists of nine shotgun and three ice-pick wounds. We agree with Willis[2] that bird-shot wounds from a sufficient distance to show a definite shot pattern on the abdomen are best treated with conservatism unless continued bleeding is suspected. Surgical repair of the many tiny puncture wounds in the bowel which almost always seal themselves, has proved needless and futile. Shotgun wounds at close range are nearly uniformly fatal because of the terrible damage produced. Ice-pick punctures, uncomplicated by severe hemorrhage, are treated because of their smallness on the same expectant rationale as shotgun wounds. Therefore, since in most of these cases it could not be proved whether or not perforation occurred, the whole group was excluded. The three patients with ice-pick wounds recovered. Three

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of the nine with shotgun injuries died. One of these, shot at close range, was moribund at entry. The other two, although showing a shot pattern, succumbed, apparently to hemorrhage and shock. These latter two probably should have been explored.

DIAGNOSIS

The presence of a gunshot or stab wound is obvious. The real difficulty that occasionally arises is whether or not the peritoneal cavity has been penetrated, for if it has, exploratory laparotomy is, with the exceptions already noted,[3] obligatory.[4-7]

TABLE I

INCIDENCE OF VOMITING AND OF SIGNIFICANT PAIN, SPASM, AND TENDERNESS

	VOMITING	PAIN	SPASM	TENDERNESS
Number of cases	19	24	27	30
Nonpenetrating	*10%*	*62%*	*22%*	*33%*
Number of cases	22	36	37	38
Penetrating s injury	*9%*	*64%*	*22%*	*37%*
Number of cases	72	129	171	150
Penetrating c injury	*46%*	*82%*	*68%*	*74%*

The unreliability of the history and physical findings in diagnosing penetration or visceral injury[7, 8, 9] is shown in Table I. It is surprising both that a greater percentage of the seriously wounded do not show significant symptoms and signs and also that these signs and symptoms are so frequently present in cases with no penetration of visceral trauma.

Penetration is obvious when protruding omentum is found. Evisceration of omentum (or rarely bowel) occurred fifty-two times among the stab wounds, an incidence of 36 per cent, and only twice in all the gunshot cases. The passage of blood by mouth, rectum, or urethra points to injury of the stomach, lower bowel, or urinary tract. However, it has been generally agreed heretofore that when the question of penetration cannot be otherwise answered, surgical exploration is indicated.[6, 7, 10, 11]

Since late 1938, we[12] have used peritoneoscopy in these doubtful cases to diagnose presence or absence of perforation (Fig. 1). We have employed this procedure, so far, fifteen times and without error. Eight times peritoneal perforation was ruled out and laparotomy avoided and in the other seven cases perforation of parietal peritoneum was disclosed and surgical exploration definitely indicated. It will be recalled that forty-six cases (13.7 per cent) were excluded from the present series because of nonpenetration, the same proportion as found by Taylor,[13] (13.8 per cent). Peritoneoscopy could have given the same information in practically every case and thus would have eliminated needless explorations.

MORTALITY ANALYSES

The total mortality for the 190 gunshot wounds is 51 per cent, for the 146 stab wounds, 14.1 per cent, combined, 32.1 per cent. The

operative mortality for the 182 gunshot cases and 145 stab wound cases that came to surgery is 48.9 per cent and 13.8 per cent respectively. These figures compare favorably with the average in the literature, which is about 57 per cent for bullet wounds and about 24 per cent for knife wounds. Fleming and McClamroch[14] report an operative mortality of 46.8 per cent for 111 perforating gunshot wounds of the abdomen. This is the lowest mortality we have found among larger series.

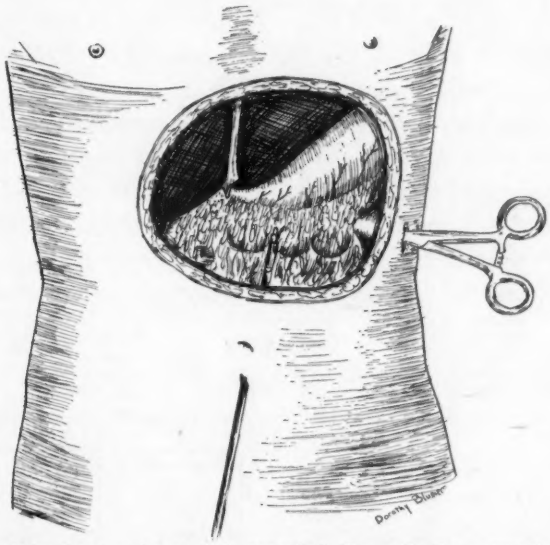

Fig. 1.—Illustrating how a hemostat, inserted through the suspected stab or gunshot wound, will indent and localize the underlying peritoneum for inspection through the peritoneoscope. (Courtesy Am. J. Surg. 54: 668, 1941.)

TABLE II

CAUSE OF DEATH

CAUSE OF DEATH	GUN		KNIFE	
	NO. CASES	MORTALITY (%)	NO. CASES	MORTALITY (%)
Shock and hemorrhage*	55	56.7	6	28.6
Peritonitis†	25	25.8	7	33.3
Pulmonary	12	12.4	1	4.8
Other or not recorded	5	5.2	7	33.3

*Fatal shock not associated with considerable hemorrhage occurred only three times, all gunshot.

†In a few of these cases pneumonia also was present.

Cause of Death.—It will be seen from Table II that by far the greatest single cause of death among the gunshot cases is shock and hemorrhage, with peritonitis less than half as frequent. As might be expected in stab injuries associated with relatively little bleeding, peritonitis from hollow viscus injury is the chief cause of death. Of the sixty-three cases of fatal shock (both gunshot and stab wound), all but three were accompanied by large hemorrhage. The preponderance of hemorrhage and shock in causing death is also pointed out by Fleming and McClamroch,[14] Loria,[18] Oberhelman and LeCount,[17] Taylor,[13] and Wilson.[6]

TABLE III

EFFECT OF AGE ON MORTALITY

AGE (YEARS)	GUN		KNIFE	
	NO. CASES	MORTALITY (%)	NO. CASES	MORTALITY (%)
0 to 20	31	45	18	0.5
21 to 40	130	47	101	11
41 to 60*	24	75	21	28.6

*There were six patients over 60 years of age, one with fatal gunshot, two with fatal and three with nonfatal stab wounds.

Effect of Age on Mortality.—It is noteworthy (Table III) that for the gunshot cases where hemorrhage plays a far greater role than in stab wounds, the big interval in mortality comes after the age of 40. This is in agreement with Rippy's[15] series and suggests that one of the reasons for the greater vulnerability of the older gunshot patients is that their arteriosclerosis prevents effective vasoconstriction, nature's hemostat. Allen and Benedict[20] explain similarly the greater hazard of bleeding duodenal ulcer in patients 50 years of age or over.

TABLE IV

EFFECT UPON MORTALITY OF TIME INTERVAL FROM INJURY TO OPERATION

TIME INTERVAL	GUN		KNIFE	
	NO. CASES	MORTALITY (%)	NO. CASES	MORTALITY (%)
2 hours or less	24	37.5	17	0
2 to 4 hours	87	51.7	62	11.2
Over 4 hours	34	50	31	16.1

Interval Between Injury and Operation.—Table IV reveals a consistent increase in stab-wound mortality as the time lengthens between injury and operation. This is to be expected since the chief cause of death in patients with stab wounds is peritonitis. The slight decline in mortality when gunshot wounds are over four hours old is undoubtedly due to the fact that hemorrhage, the largest cause of death, will have eliminated previously the worst-risk cases. The chief danger remaining for those with delayed operation will be peritonitis and pneumonia (See Table II again).

TABLE V

EFFECT ON MORTALITY OF DURATION OF OPERATION

OPERATING TIME HOURS	GUN		KNIFE	
	NO. CASES	MORTALITY (%)	NO. CASES	MORTALITY (%)
1 or less	20	55	48	8
1 to 1½	49	39	51	14
1½ to 2	54	44	26	12
2 to 2½	36	53	11	9
Over 2½	23	70	9	44

Duration of Operation.—In the case of the bullet wounds (Table V), aside from the high mortality where operating time was one hour or

less, the rate increased consistently with the increasing duration of operation. The high mortality of the briefest-operation group of gunshot wounds is largely attributable to hemorrhagic shock, which was responsible for death in 55 per cent of cases as against 48 per cent in the remaining time groups combined. Furthermore, 46 per cent of patients whose surgery lasted an hour or less, died on the table, while only 8 per cent of those with more prolonged operation did so.

The remarkably low mortality rate among stab wounds with operating time an hour or less is due to the fact that 69 per cent of them had no visceral injury.

TABLE VI

OPERATING TIME OF FATAL AND NONFATAL GUNSHOT CASES AND OPERATIVE MORTALITY IN SEVERAL SERIES*

AUTHOR	NO. CASES	AVERAGE OPERATING TIME		OPERATIVE MORTALITY (%)
		LIVED (MIN.)	DIED (MIN.)	
Oberhelman and LeCount[17]	301	72	76	61.4
Taylor[13]	101	75	85	60.0
Prey and Foster[21]	22	55	70	68.0
Hamilton and Duncan	182	102	112	48.9

*Unfortunately these are the only series with figures adapted to this comparison.

It has been urged by some[21] that great speed in operating is essential to save these patients. However, this is not borne out by Table VI. Rather, it seems that the rising mortality as the operating time increases is due, not to length of procedure per se, but to the increasing damage to be repaired. Furthermore, Rippy[15, 22] points to the increased incidence of missed visceral perforations in too-hurried operations. Unfortunately, only thirty autopsies were performed in this group, excluding cases of death on the table and nonoperated cases. Overlooked hollow visceral perforations were found nine times (30 per cent). This is below the average of the literature:

Oberhelman and LeCount[17]	55.6%	of	169	autopsies
McGowan[19]	55.5%	of	27	autopsies
Taylor[13]	43 %	of	14	autopsies
Billings and Walking[16]	19 %	of	49	autopsies
Rippy[22]	50 %	of	12	autopsies
Wilson[6]	50 %	of	20	autopsies

Some of these statistics, however, include injury to solid as well as to hollow viscera.

Mortality Related to Viscera Injured.—There is inevitably some overlapping in Table VII. Thus, if jejunum and colon were both perforated, a single entry of the more serious injury, namely of the colon, is made. Likewise, even though there may have been numerous bowel perforations in a given case, if death was obviously due to hemorrhage and shock, it is not recorded as bowel injury. If a solid and a hollow viscus were both injured and the patient died within the shock period (the first forty-eight hours) and obviously not from peritonitis or sepsis,

TABLE VII

MORTALITY OF VARIOUS VISCERAL INJURIES

VISCERA INJURED	NO. CASES	TOTAL MORTALITY (%)	MORTALITY BY BLOOD LOSS (%)	
			SMALL	LARGE
Stomach and duodenum	26	23.0	0	100.0
Jejunum	17	12.5	14.3	0
Ileum (including 5 unlocalized small bowel injuries)	24	25.0	7.1	55.5
Ileum and colon	13	23.0	10.0	66.6
Colon	40	40.0	47.0	50.0
Solid viscera	64	36.0	0	51.1
Great vessel	13	100.0	--	--
Abdomen and chest	34	60.8	--	--
Abdomen and urinary bladder	11	36.4	--	--
Abdomen and pancreas	7	71.3	--	--

death is attributed to the solid viscus injury alone. Conversely, if the death had been from peritonitis, it would be credited to the bowel injury.

The influence of large hemorrhage toward a lethal outcome in the various visceral injuries is striking. The only exception is presented by the jejunal perforations. The two fatalities in this group were associated with negligible blood loss.

Perforation of stomach and duodenum carries a rather high mortality because of the relatively large associated blood loss, the difficulty of exposure, and the frequency (73 per cent) of significant associated thoracic injuries.

There is, as we might expect, a progressive increase in mortality from peritonitis as the level of bowel perforation descends from jejunum to colon.

The apparently high mortality of associated urinary bladder perforation is deceiving, since in each of the four fatal cases the bladder injury was merely incidental in a general picture of hemorrhage and shock terminating on the operating table or within twenty-four hours.

The seven instances of pancreatic injury were associated with other more serious trauma. Four of the five fatal cases died within twenty-four hours, of obvious hemorrhagic shock. In the one autopsied case there was fat necrosis within the lesser sac, but apparently this did not contribute significantly to the fatality. The fifth patient died on the fourth day of lobar pneumonia. These findings do not corroborate the disastrous enzymatic digestion that Eisberg[23] holds will follow pancreatic injury.

The uniformly fatal outcome of all great vessel injuries is likewise reported by Loria.[18] By great vessel, we refer to femoral artery or vein, iliac artery or vein, cava or aorta.

The gravity of coincidental abdominal and chest injuries is not generally realized. The patients in these cases are in striking contrast to those admitted to our service with perforating injury of chest alone, practically all of whom recover on a conservative regime. As can be seen in Table VII, the mortality of the thirty-four cases in which there

was also significant injury above the diaphragm (moderate to severe hemothorax, pneumothorax and/or pulmonary damage) is 60.8 per cent for gunshot and stab wounds together, as against 31.9 per cent where the injury was entirely abdominal. Wilson[6] also records similar findings: 76.3 per cent mortality, chest and abdomen; 47.0 per cent abdomen alone.

Of the twenty-one fatal cases with significant thoracic injury, this injury was probably the chief lethal factor eleven times. Therapeutic interference, such as thoracentesis, was recorded in only three of these eleven cases, although such interference might have materially improved the critical condition of seven of the remaining eight patients. Of the twenty-one fatal cases, thoracentesis with or without reinfusion of hemothorax blood was performed in only seven cases (33 per cent), whereas this procedure was carried out in eight of the thirteen surviving cases (62 per cent). In one of the surviving cases, 500 c.c. of blood were aspirated and returned to the patient. In one of the fatal cases, practically moribund at the time, 2000 c.c. of blood were removed in a single aspiration from the right pleural space and returned by autotransfusion. This greatly improved the patient's blood pressure and general condition. He grew gradually weaker in spite of repeated thoracenteses and transfusions of bank blood and died the third hospital day. Too late, it was realized that his decline had probably been produced by continued active bleeding from a lacerated liver and might have been corrected by timely laparotomy. Unfortunately, an autopsy was not obtained.

Too often, preoccupation with the abdominal injuries overshadows serious and often ameliorable damage above the diaphragm. Occasionally, an initial negative chest examination or x-ray film will lull suspicions, only to have the autopsy reveal massive hemothorax or pneumothorax, or both.

TABLE VIII

EFFECT OF HEMORRHAGE ON MORTALITY

HEMORRHAGE	GUN		KNIFE	
	NO. CASES	MORTALITY (%)	NO. CASES	MORTALITY (%)
Small	79	27.8	107	9.3
Medium	39	51.3	14	14.3
Large	64	73.4	18	33.3

Effect of Blood Loss on Mortality.—Table VIII shows a striking and consistent increase in mortality with increasing hemorrhage. Blood loss reported in this paper is graded in the customary fashion:[22] Small, 500 c.c. or less; moderate, 500 c.c. to 1000 c.c.; large, 1000 c.c. or over. The tremendous influence of hemorrhage upon mortality in these perforating abdominal injuries is generally recognized (Billings and Walking,[16] Loria,[24] Mason,[25] Rippy,[15] and Taylor[13]).

SHOCK IN PERFORATING ABDOMINAL TRAUMA

In this study a patient is considered in shock whose systolic blood pressure is 80 or under. We found, as did Keckwick and co-workers,[26] that the pulse rate shows little correlation with the blood pressure and is a less accurate index of the extent of blood loss and injury. The unreliability of the blood pressure itself in this respect is shown by the thirty-four patients (Table IX) who had suffered marked blood loss with or without severe trauma and who arrived with a systolic pressure above 80.

TABLE IX

CORRELATION OF ADMISSION SYSTOLIC PRESSURES ABOVE AND BELOW SHOCK LEVEL WITH SEVERE TRAUMA, SEVERE HEMORRHAGE, BOTH OR NEITHER

SYSTOLIC BLOOD PRESSURE	SEVERE TRAUMA ALONE	SEVERE HEMORRHAGE ALONE	SEVERE HEMORRHAGE AND TRAUMA	NEITHER SEVERE HEMORRHAGE NOR TRAUMA
Above 80	0	21	13†	--
80 or lower	3*	18	16†	11

*Two of these had associated severe chest injuries.

†Six of these with systolic pressure above 80 and five below 80 had associated severe chest injury.

We are becoming increasingly convinced that for all practical purposes in perforating abdominal injury, hemorrhage and shock are synonymous. It will be observed in Table IX that only three patients whose systolic pressure upon entry was 80 mm. mercury or less had sustained severe trauma* alone. Furthermore, in two of these three instances, shock was explainable by the disturbed physiology of severe coincidental chest injury. Large hemorrhage with negligible trauma was responsible for collapse eighteen times, while large hemorrhage, together with severe trauma, were responsible sixteen times.

In eleven penetrating abdominal wounds and in three nonpenetrating stab wounds, shock was associated with neither appreciable hemorrhage nor trauma and must therefore be explained on a primary or neurogenic basis. The average pulse rate (85 per minute) of patients in primary shock, although varying widely, ranged 17 beats per minute less than that of patients in secondary or surgical shock.

In short, not only does hemorrhage play the chief role in mortality (see again, Tables II, VII, and VIII), but it seems to be the one essential cause of secondary shock. Keckwick and colleagues,[26] in their analysis of traumatic shock in British soldiers, express the belief that circulatory collapse in these cases (chiefly wounds of the extremities) is explainable entirely by the loss of blood and plasma into the damaged area. This is even more true of these abdominal injuries where bleeding into the peritoneal cavity is far more free and unlimited.

*Under "severe trauma," we have arbitrarily included such damage as ten bowel perforations or more, pulpified kidney, very extensive liver, or pancreatic injury along with other injuries, major coincidental fractures, and severe coincidental thoracic injury.

POSTOPERATIVE EVISCERATION

The incidence of wound disruption in this series is 5.6 per cent, which is extremely high. Bowen[27] and Meleney[28] place the occurrence of this complication at between 0.5 and 1.0 per cent of laparotomies in general. Taylor[13] reports an incidence of 3 per cent among his 101 cases of perforating abdominal gunshot wounds. Fortunately, the mortality of the present group, 26.3 per cent, is somewhat less than the average in the literature, namely about 30 per cent.

All but four of our nineteen eviscerations occurred between the sixth and tenth days; the shortest interval was three days, the longest nineteen. Although dietary data on these patients are not available, they are, as a rule, vigorous, well developed and well nourished and one would be less inclined to suspect them of vitamin deficiency or hypoprotenemia than the average ward patient undergoing laparotomy and in whom the incidence of evisceration is much lower. Massive hemorrhage might reasonably be expected to produce in the survivor severe hypoprotenemia. Yet in only one instance did evisceration follow severe hemorrhage, and only twice did it follow moderate hemorrhage. In the other sixteen cases the blood loss was trivial.

Of the nineteen disrupted wounds, seven were infected. Stay sutures were used in ten (no statement as to stays in five) and only one was drained. Suture material in the majority was chromic catgut, silk was used twice, and through-and-through "Kaldermic" sutures were used once.

The rectus incision was used in the combined gunshot and stab wound series 289 times with a 4.5 per cent incidence of dehiscence as against 16.6 per cent for the transverse incision, which was used eighteen times. This disparity of incidence is maintained if we consider only those cases from 1938, when we began to use the transverse incision, through 1941. During this time there have been fifty-nine rectus incisions with 3.4 per cent of eviscerations, as compared to twenty-six transverse incisions with 15.4 per cent of eviscerations.

Why the incidence of wound disruptions is so high among these cases and especially so among those explored through a transverse incision is an unsolved problem. Perhaps part of the answer is mechanical stress and strain. Many of these patients are intoxicated and uncooperative, often kept in bed only by dint of restraints.

TREATMENT

Time of Operation.—All are agreed that any abdominal injury proved to be penetrating, with the exception of bird-shot or ice-pick wounds, should be explored promptly, but opinion is divided as to the advisability of delaying operation to treat shock. Meyer and Shapiro,[3] Wilson,[6] Storck,[9] Billings and Walking,[16] Loria,[24] and Rippy,[15, 22] advise against operating, if possible until the shocked patient's blood pressure has risen above the critical level. Fleming and McClamroch[14] and Van

Schaick[29] believe that since shock in these injuries is nearly always due to hemorrhage, nothing is to be gained by delaying operation for shock therapy and they recommend that antishock measures be conducted along with surgical intervention. We agree with the latter authors, for we fail to see how blood dribbling into a vein through a needle can compensate for what is meanwhile pouring out into the abdomen through torn vessels.

However, teamwork is important; antishock measures, such as the rapid administration of blood and plasma[26] from the blood bank should not wait upon surgery, but should proceed with it. Thus, by the time the peritoneum is opened a pint of plasma should be well on its way into the vein.

TABLE X

OUTCOME OF OPERATION UPON PATIENTS STILL IN SHOCK
(SYSTOLIC BLOOD PRESSURE 80 OR LOWER)

		SEVERE HEMORRHAGE ALONE	SEVERE TRAUMA ALONE	BOTH	NEITHER	NOT SPECIFIED	TOTAL
Came out of shock during operation	Lived	5	0	1	10	1	27
	Died	2	0	4	2	2	
Remained in shock throughout operation	Lived	0	0	2	0	0	13
	Died	2	0	8	0	1	

The outcome of patients on whom operation was begun while their systolic pressure was 80 or under (Table X), seems to justify immediate surgery in these patients in whom shock is present. Of the forty cases with available data, only thirteen failed to recover from hypotension in the course of the operation. Furthermore, none of the twelve patients in apparent primary or neurogenic collapse (recorded in Table X as having "neither" severe hemorrhage nor severe trauma) failed to come out of that state during the course of the operation. In this primary shock group, the two deaths, due to bronchopneumonia, could scarcely be ascribed to their having undergone operation while still in collapse, especially as they recovered from it during the procedure. We believe it a fallacy, then, to delay operation for the treatment of secondary shock when operation is the one way of controlling hemorrhage, its cause. On the other hand, operation, gently performed and with intravenous fluids running, will not appreciably aggravate benign or neurogenic shock.

Transfusion and Autotransfusion.—The value of transfusion in these cases no longer needs emphasis. Patients in shock or in whom active bleeding is likely, should from the start receive infusions of plasma or blood in so far as it is available in preference to saline and glucose solutions, since the depleted circulation may be dangerously diluted and further plasma proteins lost by too liberal use of the latter (Minot and Blalock[30]).

Furthermore, we feel that blood or plasma should not be withheld preoperatively on the grounds that by raising the patient's pressure it will promote further bleeding. Analysis of the effectiveness of transfusions among our cases is misleading. Of the seriously injured patients, the group that died averaged 725 c.c. blood per patient, while those that lived received 600 c.c. each. Obviously, however, the most desperately wounded died in spite of the larger amount of blood received. Rippy[15] gives the mortality of his large hemorrhage group receiving transfusions as 74.4 per cent, as against 90.7 per cent for those not transfused.

Autotransfusion, however, has offered brilliant possibilities since 1935, when our surgical service adopted the policy of returning to the seriously injured patient all free peritoneal blood, contaminated by bowel contents or otherwise, that was not over six hours old. The rationale of this has two simple bases, first the saying familiar to all, "You can't infect the blood stream" and second, "Save this patient now and worry about possible infection later." It is surprising that this practice was not adopted years ago. Meyer and Shapiro,[3] Storck,[9] Billings and Walking,[16] Mason,[31] and others recommend auto transfusion only if it is certain that there is no bowel injury. Trimble[32] considers liver injury a contraindication of the procedure, on the basis of possible toxic autolysates. Van Schaick[29] strongly advocates autotransfusion and reports returning 450 c.c. of blood to a woman with a gunshot perforation of the stomach. This is the only instance we are familiar with in which contaminated blood has been reinfused into the patient.

In an analysis of all autotransfusions given at the Louisville City Hospital, inclusive of those for abdominal injuries, Griswold and Ortner[33] found but one instance in which autotransfusion of contaminated blood appeared responsible for death. In this patient, who had received numerous gunshot perforations of the bowel, autopsy revealed multiple small pulmonary emboli. Due to a technical error the reinfused blood on this occasion had been imperfectly filtered. The great majority of autotransfused patients show little if any deviation from the expected postoperative course.

The technique of autotransfusion is simple[33]: The free blood is aspirated into a citrate-containing trap bottle. This citrated blood is poured through ten thicknesses of gauze into an intravenous outfit for retransfusion.

TABLE XI

AUTOTRANSFUSIONS

AMOUNT GIVEN (C.C.)	HOLLOW VISCERA INJURED		SOLID VISCERA INJURED*	
	LIVED	DIED	LIVED	DIED
500† to 1000	2	5	6	1
1000 to 2000	2	7	3	1
Over 2000	0	3	1	1

*In 3 cases omentum or mesentery, and not a solid viscus, was injured.
†Once 300 c.c., and once 400 c.c. blood were given.

The thirty-two autotransfusions given in this series are set forth in Table XI. In all but three of the fatal cases death was obviously due to shock and hemorrhage and occurred either on the operating table or within twenty-four hours. The following cases with entirely successful outcome illustrate what a lifesaving reservoir contaminated intraperitoneal blood may be:

Lacerations of liver	autotransfused 2000 c.c.
Lacerations of stomach and liver	autotransfused 800 c.c.
Ileum perforated 4 times	autotransfused 1500 c.c.
Ileum perforated 12 times	autotransfused 1200 c.c.

We urge that this relatively safe and immediately available source of blood be no longer withheld from critically wounded and exsanguinated patients. To be sure, autotransfusion will not be a necessity where gallons of plasma and blood are available, but how often is this the case either in civil practice or among war casualties?

Auxiliary Procedures.—The x-ray and fluoroscope play an important diagnostic role. If the path of a bullet with entry wound only is in doubt, the slug can be localized roentgenologically as the patient is en route to the operating room. The progress of concomitant thoracic injury should be studied by an initial film and later, if necessary, by repeated bedside plates.

Oxygen[34] is administered to patients in shock, either by intranasal catheter or by tent, to combat anoxemia.

Wangensteen suction for decompression of the upper gastrointestinal tract has become a routine for all patients with injury to stomach or bowel. The Miller-Abbott tube is used in case of distention or ileus.

In all cases of peritoneal soiling 10 Gm. of sulfanilamide powder are placed in the abdominal cavity and another 3 to 5 Gm. in the operative incision. More recently, trial is also being made of sulfathiazole and sulfadiazine.

Adrenal cortical extract[35] is probably of some value in combating shock. The synthetic cortical extract desoxycorticosterone acetate[36] since it requires at least six hours to reach its maximal efficiency is of less value in established shock. Our experience with this sterone preoperatively in about fifty major operations has shown no striking prophylaxis against abnormal hypotension and tachycardia when compared to an equal number of control cases. However, our final analysis of this study has not been completed.

Anesthesia.—Local anesthesia should not be used in these cases. It is time consuming and must be supplemented in nearly all instances, since adequate exposure and free manipulation of viscera is essential. In the present series spinal anesthesia was employed 165 times with a mortality of 25 per cent and general anesthesia, 151 times, with a mortality of 43 per cent. Since general anesthesia was reserved for the low blood pressure group, which was also the more gravely wounded, this disparity in death rates is not surprising. We feel that the greater relaxation secured by spinal anesthesia more than compensates for its theoretical disadvantage, namely that it stimulates peristalsis and thereby allows

fecal spillage. It is therefore the anesthetic of choice unless contraindicated by low blood pressure.

Technical Details.—Thorough discussions of operative principles and details have been presented by Meyer and Shapiro,[3] Storck,[9] and others. Only a few points will be emphasized here.

Abdominothoracic injuries have already been discussed. To avoid overlooking serious intrathoracic injury, repeated portable plates and exploratory thoracenteses should be done in such patients growing worse out of proportion to their abdominal injuries. Aspirated blood not over six to eight hours old should be reinfused into the patient. Tension pneumothorax requires catheter drainage and water trap. If active bleeding continues or the pneumothorax cannot be controlled, thoracotomy should be considered. An initial transthoracic approach is recommended by Gordon-Taylor[37] where the path of bullet or shrapnel crosses the upper abdomen and diaphragm, especially on the left side, a zone which he terms "that fatal left subphrenic area of the abdomen." We have had no experience with this approach but it might prove valuable when significant chest injury as well as upper abdominal injury is suspected at the outset. After the chest lesions are dealt with, the left upper abdomen can be readily explored through the diaphragm.

Abdominal injuries should be approached through an unusually long incision that will allow maximal exposure. We are employing with increasing frequency, the transverse rectus-dividing incision as described by Gurd[38] because its better exposure more than compensates for the high incidence of evisceration. This latter disadvantage we hope to overcome by closing with through-and-through "Kaldermic" or braided silk sutures.

Hemorrhage is to be controlled first and thereafter bowel perforations sutured. Location of bleeders and visceral injuries will be greatly simplified by following the estimated track of the bullet or knife from the entry point in the peritoneum.

It will be found helpful in dealing with active hemorrhage, to apply temporary digital pressure proximally, i.e., compression of the hepatic artery for bleeding from the liver, of the mesenteric or lienal arteries for bleeding from mesentery or spleen, etc.

In the routine examination of the intestine, time and needless manipulation will be avoided by placing a Babcock forceps on the bowel for a marker and "reading" in both directions. Evisceration of more than one loop of bowel at a time during inspection is to be avoided if possible, because of the chilling effect and the drag on the mesentery. A thorough exploration of stomach and bowel is well worth the time spent as it will be recalled that in from 30 to 50 per cent of autopsies, overlooked visceral perforations are found.

If the posterior of two gastric perforations cannot be brought into view through the gastrocolic or gastrohepatic omenta, free enlargement of the anterior perforation will allow satisfactory transgastric exposure and repair (Prey and Foster[21]).

The kidney region is quickly and easily exposed by incising the avascular lateral colic reflection of peritoneum and reflecting colon and its mesentery mesially.

We agree with Storck[9] that enterostomy proximal to sutured perforations or anastomosis is futile. Satisfactory bowel decompression can be obtained with Wangensteen suction or Miller-Abbott tube.

Abdominal drainage is in most cases useless. However, the kidney fossa and retroperitoneal tissues, if soiled with lower bowel contents, should be drained through a stab wound in the flank. A drain should also be placed if there has been wounding of pancreas or biliary tract, to provide escape for possible leakage of pancreatic juice or bile.

SUMMARY

1. One hundred ninety perforating gunshot and 146 perforating stab wounds of the abdomen have been reviewed.

2. The mortality for the entire series is 51 per cent for the bullet wounds and 14.1 per cent for the stab wounds. The operative mortality is 48.9 and 13.8 per cent respectively.

3. These observations and analyses are made:

a. Incidence of vomiting, pain, spasm, and tenderness in nonpenetrating as against penetrating injury.

b. The chief causes of death.

c. The effect on mortality of age, time from injury to operation, duration of operation, and amount of blood lost.

d. The various visceral injuries and their mortality.

e. The cause of shock in perforating abdominal injuries. (Primary and secondary shock are differentiated.)

f. The outcome of patients operated upon while still in shock.

g. Postoperative eviscerations.

4. The following points are particularly emphasized:

a. The value of peritoneoscopy in diagnosing peritoneal perforation in doubtful cases.

b. Hemorrhage is synonymous with shock in perforating abdominal trauma and therefore operation to control hemorrhage brooks no delay.

c. Abdominothoracic injury is more serious than generally realized and ameliorable chest injuries are too frequently overlooked or inadequately treated.

d. Autotransfusion, regardless of blood contamination, is urged for all seriously injured patients operated upon within six hours.

5. Treatment is outlined and a few technical details are discussed.

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CONSERVATISM IN THE SURGICAL MANAGEMENT OF ACUTE REGIONAL ENTERITIS

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MUCH has been written in the last few years about regional enteritis and much has been accomplished toward clarifying its puzzling aspects since its original description by Crohn and his associates[1] in 1932. With regard to the acute phase of the disease, surgical treatment roughly divides itself into radical and conservative. The following case reports and discussion of the literature are offered in support of the view that radical treatment is not indicated in those cases of acute regional enteritis initially encountered at laparotomy for an acute abdominal condition. Emphasis is placed upon the fact that the cases and discussion presented below represent that phase of regional granulomatous inflammation of the bowel which is acute or subacute when first seen and which presents clinical findings demanding celiotomy at the time of admission to the hospital.

CASE REPORTS

CASE 1 (Roper Hospital No. 81503).—A 23-year-old colored male, a laborer, was admitted May 18, 1935, with a history of generalized lower abdominal pain, more pronounced in the right lower quadrant, of three days' duration. The pain was intermittent in character and was accompanied by nausea at the onset. Vomiting occurred three times during the first thirty-six hours and then stopped. The patient took castor oil at the onset of symptoms and had a watery diarrhea for the next forty-eight hours. When he reached the hospital, the discomfort of the patient was entirely confined to the right lower quadrant. There had been no urinary, respiratory, or cardiovascular disturbances at any time. The past and family histories were irrelevant. Examination on admission revealed a well-developed negro adult male, obviously acutely ill. The abdomen was flat but did not move with respiration. There was generalized lower abdominal tenderness on palpation which was excruciating over McBurney's point and just below the umbilicus. Rebound pain from left to right was well defined. The right rectus muscle was frankly rigid in its lower half. No masses were palpated. By rectal examination, acute tenderness and suggestive induration were noted high in the right pelvic fossa. Auscultation revealed active peristalsis to be present. Temperature was 102.6° F.; blood pressure 124/66; leucocyte count, 9,950, with 71 per cent polymorphonuclear cells; urinalysis was normal. A diagnosis of acute appendicitis was made.

Operation.—Under spinal anesthesia, the abdomen was opened through a McBurney incision. A large amount of free serosanguineous fluid was present in the abdominal cavity. Exposure through the gridiron opening was found to be inadequate and a right rectus muscle-splitting incision was made. The appendix was normal and was firmly adherent to the ileocecal region by old fibrous bands. The terminal six inches of the ileum were intensely edematous and hyperemic and

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the lumen was almost completely occluded. There was a regional lymph node hyperplasia and the adjacent mesentery showed small areas of fresh granulation tissue. No other abnormalities were noted. The appendix was removed and both incisions were closed in layers.

Culture of the peritoneal fluid was sterile. Culture of feces following the operation revealed normal intestinal flora. The pathologist's report of the appendix was "no abnormality."

The postoperative course was mild. On the fifth day the temperature became normal and the patient was given a soft diet. He was discharged symptom-free on the fourteenth postoperative day.

This patient has been seen on numerous occasions since operation and at no time has he complained of abdominal discomfort of any kind. An x-ray of his abdomen following barium enema in July, 1941, showed the ileocecal valve to be fully patent and the terminal ileum to be well filled. There was no "string sign." This latest checkup was six years and two months after operation. The patient is now considered to be cured.

CASE 2 (Roper Hospital No. 104297).—A 39-year-old colored male, a farm-hand, was admitted to the hospital May 26, 1939, complaining of intermittent left lower quadrant pain of three days' duration. The onset was extremely sudden and the pain of a sharp lancinating nature. Prior to the onset of symptoms, constipation was present and only one bowel movement occurred in five days. The patient took castor oil about three hours following onset of acute seizure and promptly vomited. He continued to vomit for twenty-four hours, then suddenly stopped, at which time a diarrhea appeared and persisted until admission to the hospital. Meanwhile, the left lower quadrant pain persisted without radiation. There were no urinary, cardiovascular, or respiratory disturbances. Past history was irrelevant except for frequent headaches and intermittent nocturia and frequency, thought to be the result of numerous gonorrheal infections. Examination on admission showed a well-nourished negro male to be acutely ill and writhing in pain. Blood pressure was 109/69, temperature, 98° F., and pulse 100 per minute. The abdomen was not distended and moved freely with respiration. Palpation caused exquisite pain over the entire left portion of the abdomen, most marked about 1½ inches below and to the left of the umbilicus. There were no masses. Peristalsis was not audible and rectal examination contributed nothing of diagnostic value. The leucocyte count was 10,100 with 85 per cent polymorphonuclears. Urinalysis showed nothing abnormal. No specific diagnosis was made, but exploratory laparotomy was thought advisable.

Operation.—The abdomen was opened through a left midrectus incision, using spinal anesthesia. There was no appreciable increase in free peritoneal fluid. The proximal four inches of the sigmoid were heavily thickened and indurated and there was a diffuse vascular engorgement of the serosa. Numerous organizing adhesions plastered the sigmoid colon against its mesentery. No evidence of diverticulitis was found. Regional lymph node enlargement was noted. The appendix and remainder of the alimentary canal were normal. Release of the sigmoid by freeing its adhesions was effected, following which the appendix was removed. One enlarged lymph node was removed for study and the abdomen closed in layers. Pathologic diagnosis from sections of the lymph node was chronic lymphadenitis, while the appendix was reported as showing fibrosis.

Recovery of the patient after operation was prompt. The wound healed per primam. On the twelfth postoperative day, an x-ray with the aid of a barium meal revealed the colon to be well filled and showed no abnormalities in the sigmoid region. The patient was discharged symptom-free on the thirteenth postoperative day.

Follow-up studies of this case have shown the patient to continue in an excellent state of health for two years and two months. A barium enema in July, 1941,

revealed the sigmoid colon to be normal throughout, showing no spasticity or "string sign" and no diverticula.

CASE 3 (Roper Hospital No. 110155).—A 21-year-old colored male, a hotel bellhop, entered the hospital April 19, 1940, complaining of pain in the right lower abdominal quadrant. Forty-eight hours before admission the patient developed a dull gnawing ache in the right half of the abdomen and in the epigastrium. The pain grew steadily worse and vomiting occurred twice shortly after its onset. About twelve hours prior to admission, all discomfort became localized in the right lower quadrant and persisted in that area, undergoing exacerbations and remissions but never completely disappearing. One bowel movement had occurred the day before admission and was described as normal. There was no diarrhea. Nocturia of moderate degree had been present for several weeks and for three days before entering the hospital the patient suffered some burning on urination unaccompanied by hematuria, dysuria, or passage of stones. The history of the systems was irrelevant and past history totally noncontributory. Examination of the patient showed a well-developed colored youth in obvious distress but not acutely ill. Temperature was 100° F., pulse, 92 per minute, blood pressure, 117/70. The abdomen was flat and held motionless during respiration. No abnormalities of the left abdominal quadrants were noted, while the right rectus muscle was rigid and acute tenderness was elicited over McBurney's point. Rebound tenderness was referred from the left lower quadrant to the midline just below the umbilicus. Rectal palpation elicited sharp pain in the right pelvic fossa; there was no mass. The white cell count was 15,000 with 64 per cent neutrophiles. The urinalysis was normal. A diagnosis of acute appendicitis seemed inescapable.

Operation.—The abdomen was opened through a McBurney incision under ether anesthesia. The appendix appeared innocent and there was considerable free clear peritoneal fluid present. The ileum in its terminal four inches was thickened and intensely hyperemic. Its consistency was boggy. The ileocecal valve was patent but definitely narrowed. There was no involvement of the cecum. A regional lymphadenopathy was present and marked. Culture from the peritoneal fluid was made. A lymph node was excised for study and the appendix was removed followed by inversion of its stump. Pathologic diagnosis from sections of the node was chronic lymphadenitis, etiology undetermined; the removed appendix showed fibrosis only on microscopic study. The peritoneal culture was sterile.

Convalescence of the patient was stormy. He suffered for several days from a watery diarrhea and intermittent, cramping, generalized abdominal pain. Cultures of stools showed normal intestinal flora; no streptococci or dysentery organisms were discovered. A tuberculin test was negative. Gradually, the symptoms subsided and a soft diet was tolerated on the ninth day postoperatively. The wound healed per primam and the patient was discharged on the twenty-first postoperative day. Six weeks after the operation, a barium x-ray showed normal patency of the ileum and the ileocecal valve but there was considerable irritability of the terminal ileum.

Repeated checkup visits of this patient found him in an excellent state of health and he has offered no complaints of any kind. In August, 1941, barium enema and x-ray revealed the terminal ileum to fill normally; there was no encroachment upon its lumen. The patient is considered well fifteen months after operation.

CASE 4 (Roper Hospital No. 116545).—This case is of too recent origin to be considered a spontaneous cure. It is offered, however, to emphasize the necessity of conservative surgical treatment in that smaller group of patients who present massive involvement of the bowel and so-called "skip areas" (see Discussion).

A 21-year-old white male, a meat packer, entered the hospital April 16, 1941, with the chief complaint of pain in the right lower abdominal quadrant, of one

month's duration. Pain was said to have been associated with frequent nausea and epigastric fullness but was not accompanied by vomiting. Constipation had been present for some months and there was a marked anorexia. Undue fatigue and slight weight loss developed rather abruptly two weeks before admission. There was no abnormality in the history of the systems. Past and family histories failed to add pertinent data. On physical examination, the temperature was 99.6° F., pulse 85 per minute, and blood pressure 120/80. The abdomen was essentially normal except for pain on deep palpation over McBurney's point. Rectal palpation was normal. The leucocyte count was 14,000 with 78 per cent polymorphonuclears and 8 per cent eosinophiles. Urinalysis was normal except for 1 plus albumin.

A tentative diagnosis of intestinal parasites was made in view of the patient's occupation and the eosinophilia. Several stool examinations, some by special laboratory technique, failed to reveal the presence of parasites or ova. The eosinophilia varied from 6 to 11 per cent. A barium meal and x-ray showed normal stomach and duodenum but a large diverticulum of the jejunum, which retained its barium, was noted. This radiographic study was repeated and similar results obtained. X-rays of the colon after a barium enema and of the thoracicolumbar spine were normal. A flat plate of the genitourinary tract was normal.

After the medical department had failed to demonstrate a cause for the eosinophilia, a presumptive diagnosis of jejunal diverticulitis was made and celiotomy advised.

Operation.—Using gas-ether anesthesia, a right paramedian incision was made. There was no increase in peritoneal fluid. The jejunum, from the ligament of Treitz to its junction with the ileum, was injected and greatly thickened. Its consistency was soft and boggy. The lumenal diameter was reduced. There was a widespread mesenteric lymphadenitis present which was confined to the jejunal mesentery. No abnormality of the other abdominal viscera was noted. The appendix was removed and a lymph node was obtained from the jejunal mesentery for study. The pathologic report from sections of the node was hyperplastic lymphadenitis, and from sections of the removed appendix, no definite abnormality.

The postoperative convalescence on the whole was smooth, but was complicated by separation of the skin edges of the incision after removal of the sutures. Secondary closure healed well. A barium meal was administered on the seventeenth postoperative day and the x-ray showed some clumping of barium in the jejunum but no evidence of stricture was seen. The patient was discharged on the twenty-third day postoperatively. Three recent follow-up visits have found this patient to be rather lethargic and weak. He complains of constant nausea and anorexia but a recent barium radiograph four months after his operation was normal, indicating no progression of the jejunitis toward obstruction or fistula formation. His temperature remains normal and a white blood count made at the time of the last x-ray was 7,500 leucocytes with a normal eosinophile count.

CASE 5 (Roper Hospital No. 118223).—Report of this case is included only to demonstrate the fact that a strong tendency toward spontaneous healing must exist in certain cases of regional enteritis. The case is of too recent origin to be considered cured (see Discussion).

A 27-year-old colored female entered the hospital July 12, 1941, complaining of severe pain in the right lower abdominal quadrant of some twenty hours' duration. At onset, the pain was located in the epigastrium but became localized in the lower abdomen after a few hours. Nausea was pronounced, but no vomiting occurred. One bowel movement early in the illness was effected with the aid of an enema. There were no other symptoms of consequence and the history of the systems was irrelevant. A similar episode was said to have occurred one month previously but disappeared after twelve hours without treatment. Examination revealed a temperature of

100° F., pulse 85 per minute, and blood pressure 110/60. The abdomen was flat and failed to move with respiration. Exquisite tenderness was present over the entire right lower quadrant making accurate palpation of deep structures impossible. Rebound pain was intense. Rectal and vaginal examinations elicited extreme tenderness in the right pelvic fossa but no mass could be demonstrated. The leucocyte count was 15,200 with 84% polymorphonuclears. Urinalysis was essentially normal. The diagnosis of acute suppurative appendicitis, possibly with rupture, appeared obvious and operation was advised.

Operation.—Under spinal anesthesia, the abdomen was opened through a McBurney incision which later had to be extended downward along the right rectus sheath. There was a large amount of dirty, brown, thin fluid free in the abdominal cavity. The cecum, appendix, distal six inches of the ileum, and a large wad of mesentery were inextricably bound together in one large, friable, fixed inflammatory mass which was covered with new granulation tissue and fibrin. On dissecting the mass free, the appendix was noted to be normal while the terminal ileum was bright red, thickened, and covered with granulation tissue. The cecum and proximal three inches of ascending colon closely resembled the terminal ileum and a spontaneous perforation of the cecum one inch in diameter had occurred adjacent to the base of the appendix. The opening had been effectively sealed off within the inflammatory mass. Regional hyperplasia of mesenteric lymph nodes was marked. Further dissection of the friable structures unfortunately resulted in tearing the cecal perforation into a rent of alarming dimensions. Very little gross soiling occurred, however. The appendix was removed and the cecal tear closed with three layers of chromic 0 catgut. Serosalization of the entire field was impossible due to the marked friability of the tissues. Generous portions of necrotic serosa and fresh granulation tissue were obtained for biopsy study. Sulfanilamide crystals, 10 Gm., were dusted throughout the right lower abdominal quadrant. Penrose drains were placed in the cecal pouch and right pelvic gutter. Closure of the incision was effected in layers with interrupted sutures.

A culture of the peritoneal fluid was negative. Biopsy studies revealed granulomatous inflammatory tissue from the intestine and appendix but failed to uncover any specific etiologic agent. No evidence of tuberculosis was seen in the sections. The appendix showed, on section study, no disease of its mucosa but heavy serosal infiltration by a nonspecific chronic inflammatory process.

The patient's postoperative course was satisfactory in every way. The drains were removed on the third postoperative day and soft diet allowed on the fifth day. The wound healed well except for a small sinus persisting at the point of exit of the drains. The patient was discharged on the fourteenth day after operation. The wound sinus healed rapidly. On Sept. 3, 1941, the patient was discharged from the outpatient department symptom-free and apparently in excellent health. She is to return at intervals for radiographic study.

DISCUSSION

A critical evaluation of the foregoing case reports brings to mind the question of correctness in diagnosis. Clinically, a preoperative diagnosis of acute regional enteritis in a patient seen shortly after the first attack of pain is purely tentative; in the majority of instances the condition is not mentioned among the diagnostic possibilities. Only when celiotomy is performed and direct inspection and palpation of the affected bowel is allowed can one specifically state the existence of acute regional enteritis. Radiographic study of the bowel with the aid of barium is of only limited value in verifying the presence of the disease, in that the acute cases which subside spontaneously give no positive

roentgen findings during the acute phase other than irritability of the involved segment. The so-called "string sign" belongs to the chronic phase of regional enteritis and makes its appearance quite late in the disease, being dependant upon narrowing of the bowel lumen due to actual fibrosis of the bowel wall. Thus, all x-ray studies of the foregoing cases have been noncontributory as far as verification of the diagnosis is concerned inasmuch as all examinations were made during subsidence of the disease and following recovery. The diagnosis in each instance has been established only after the combined expedients of direct inspection and laboratory exclusion studies.

In reviewing the literature on regional enteritis since its introduction in 1932, one is confronted by the fact that after nine years of study there remains a distinct division of surgical opinion regarding treatment of the acute phase of the disease. Since 1938, however, there appears to have been a definite trend toward conservative management, although proponents of radical measures (enterostomy, resection and short-circuiting procedures) still exist.[2, 3, 4]

The recommendation of conservative treatment (which implies simple exploration with or without appendectomy) must be prefaced by consideration of certain pathologic and clinical features of the disease. To begin with, the etiology of acute regional enteritis is not known. It seems logical to assume that radical manipulation of an acutely inflamed segment of bowel might kindle a smouldering fire into the flaming proportions of peritonitis, even though extensive resection were accomplished. Such a situation is definitely undesirable in a process whose mode of onset and manner of progression are not understood. On the other hand, should the acute disease progress to a chronic state, the treatment of which is undeniably radical resection, the patient can be kept under observation and prepared in advance for an extensive surgical procedure. That progression of the acute state occurs in some instances is well established[4, 11, 12] and the responsibility for its detection is placed directly upon the surgeon who handles his acute cases by conservative methods. Thus, the conservatist is dutifully bound to adopt an organized plan for observation and periodic follow-up examinations, with particular stress upon repeated barium x-ray studies, in all patients treated by laparotomy alone. Proper preoperative management by transfusions, general supportive measures, and some form of enteral chemotherapy, such as sulfanilylguanidine[5, 6] or succinyl sulfathiazole,[34] should take precedence over the choice of radical treatment in an unprepared patient just recently admitted to the hospital. This becomes especially true when one considers that the mortality rate in resection, even under elective conditions, is formidable and that recurrences after resection are reported, varying from Crohn's[8] minimal estimate of 7.7 per cent to as high as 15 per cent.[9]

Spontaneous subsidence of acute regional enteritis, "possibly never to be heard from again,"[8] must be of frequent occurrence. Actual

visualization at the operating table of normal bowel previously diseased is reported by Pessagno.[10] Case 5 of this series indicates beyond doubt that a strong healing tendency must exist in some instances of acute regional enteritis, since primary suture of a perforated cecum in the presence of an extensive process brought about healing without fistula formation. Relative proof of the spontaneous disappearance of the acute disease can be had in radiologic study of the bowel at intervals after operation (Cases 1, 2, and 3 of this series). Beyond the phase of x-ray study, one is left only with the patient's symptomatic progress as a guide to evaluating the degree of activity of the disease. The literature offers many reports of spontaneous cure after simple exploration, with or without appendectomy.[13-26] Many of these reports embody case studies representing freedom from symptoms for many years; some include roentgenologic findings. Especially striking are those patients who have remained symptomless for a period of years. Of seven cases investigated by Lehman,[20] two had had no recurrence nine and one-half years after operation (appendectomy) while a third was well after four years. Brown,[27] Rockey,[13] Rixford,[15] Kross,[23] Eliason and Johnson,[22] and Eckel and Ogilvie[24] all have contributed studies of patients, followed for at least four years, who have remained well after conservative treatment. In an all-inclusive review of the world literature, Ravdin and Johnston[28] concluded from their own experience and reports of other authors that extensive surgical procedures are not indicated in acute regional enteritis. They emphasize the fact that a large number of cases subside spontaneously and that in those few which progress to a chronic state, as noted above, careful preoperative management is essential to good results in later resection of the bowel. Direct recommendation for conservative surgical treatment in the acute phase is made by several authors whose writings reflect surgical experience of unusual scope.[29-32] It is interesting to note that Mixter's[12, 32] wide experience has led to a change in his attitude from radical to conservative between the years 1935 and 1939.

There are noted occasionally anatomic features of distribution in certain cases of acute regional enteritis which literally demand that only the simplest surgical methods be employed. In case 4 above, the entire jejunum was found involved at operation, so that radical surgical management was out of the question. Similarly, the occurrence of "skip areas" throughout the intestinal tract provides technical difficulties in resection or short-circuiting which defy the boldest operator. Furthermore, the overenthusiastic surgeon may find that, after removal of an acutely inflamed segment of bowel, a similar acute process exists at a higher level of the alimentary canal. Pitfalls of this nature are eliminated when simple exploration, appendectomy, and closure of the wound are accomplished, to be followed by watchful waiting.

Since removal of the appendix in the presence of an acute granulomatous condition of the intestine has occasioned considerable comment,

brief mention of certain of its aspects is included here. Emphasis is given to the fact that appendectomy in acute regional enteritis is purely incidental and not designed to alter the course of the disease per se. Tendency to fistula formation is thought by some to be increased by appendectomy in the acute phase. Certainly, the incidence of such is quite low if one depends upon the literature as a gauge. In all cases reported in this series, appendectomy was done, and in Cases 1 and 3 the site of the disease was the terminal ileum, while in Case 5 actual extension to the cecum was present; no fistula resulted in any case. Eliason and Johnson[22] offer the assumption that if the appendix were not removed and later became involved, appendical obstruction due to edema could conceivably take place and early rupture occur. Fallis[33] and Lehman[20] report seven and six cases, respectively, of acute terminal ileitis treated by simple exploration and appendectomy; no fistula occurred.

SUMMARY

1. Five cases of acute regional enteritis are reviewed. All were managed by simple exploration and appendectomy and none showed progression of the disease.

2. That a strong tendency toward spontaneous healing is present in certain instances of acute regional enteritis is illustrated by Case 5, in which prompt healing followed primary closure of a perforated cecum in the presence of a very active acute process.

3. Conservative management is mechanically essential in a smaller group of acute cases showing either massive involvement of the bowel or extensive "skip areas."

4. Spontaneous disappearance of acute regional enteritis occurs in a large number of instances, progression to the chronic phase in few.

5. Recurrence after radical resection is relatively frequent, not to mention a formidable mortality rate in patients subjected to radical surgical treatment during the acute phase.

6. In order to detect the occasional case showing a tendency to progress to chronic regional enteritis, conscientious observation and frequent postoperative x-ray study are essential in the acute case managed by simple exploration and appendectomy.

7. Progression from acute to chronic under observation and thorough preoperative preparation by general supportive measures, transfusions, and enteral chemotherapy constitute a far more desirable form of management than radical resection in an unprepared acute case just recently admitted to the hospital.

8. Appendectomy is incidental in the conservative form of surgical treatment; it does not materially increase the risk of fistula formation.

9. If the appendix is not removed at operation, eventual involvement of it by the acute granuloma may cause appendical obstruction from edema with consequent early rupture and peritonitis.

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OPERATIVE CHOLANGIOGRAPHY

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CHOLANGIOGRAPHY DURING OPERATION

ONE of the chief problems of gall bladder surgery has to do with exploration of the common duct. If no stones are in the common duct, it is best to leave it alone; if stones are present, it is essential to remove *all* of them. Every surgeon of large experience knows that it is often difficult to tell whether stones are present and even more difficult at times to tell whether all stones present have been removed.

Our criteria for internal exploration of the common duct during the operation have been (1) patient jaundiced at time of operation, or a history of jaundice; (2) palpable stones in common duct; (3) dilated common duct.

With these criteria we find that in our last 500 cholecystectomies we have opened the common duct forty-five times or 15 per cent. In about one-third of these instances no stones were found, that is, in those cases it would have been better had we let the common duct alone. We do not think debris or so-called "sand" which can be scooped out of any common duct when the gall bladder itself contains "sand" has any special significance or requires mechanical removal. Experience has shown us again and again that the fact that a probe can be passed with ease through the common duct into the duodenum is not an absolute sign that no stone is present in the duct. In a dilated common duct a stone of large size can lie in the lumen and not come in contact with the probe at all as it makes its way through the duct. Even when the duodenum is mobilized, a procedure we use with great frequency, the stone may not be palpable without or with a probe in the duct.

In one instance that we know of for a certainty we did not remove all of the stones which were present in the common duct but left one behind. The patient in question died of a pulmonary embolus on the tenth day of an apparently excellent postoperative convalescence. At autopsy the overlooked stone was found lying free in the common duct. We had exercised every possible precaution at the time of operation, had palpated the duct inside and out, had flushed out the duct, passed probes through the duct into the duodenum and had probed up into the hepatic ducts, yet here at autopsy was the stone the size of a large pea, mute but absolute evidence that the methods at our command were inadequate for obtaining

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an accurate knowledge of whether or not stones were present in the duct. In the hope of reducing, if not completely eradicating, the possibility of repeating such a mistake we have for the last two years made use of cholangiography during operation wherever we thought indicated. As we have become more familiar with this procedure our indications have increased and we now use immediate cholangiography whenever we feel that there is the slightest doubt that a common duct stone may be present.

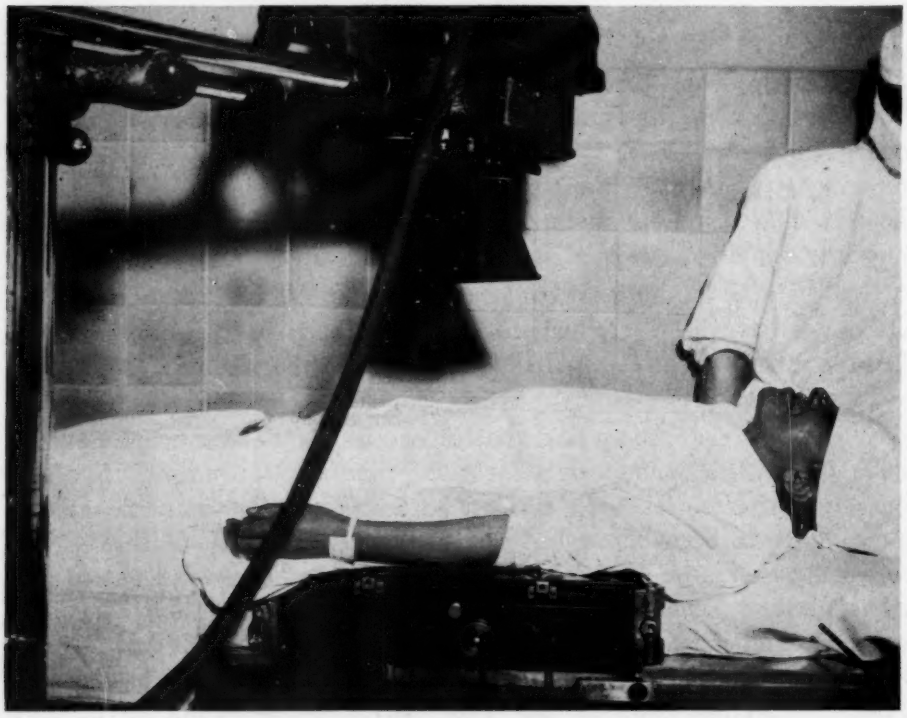

Fig. 1.—Method of placing patient on table. Note the Bucky diaphragm and the pads above and below so that the patient lies comfortably on a plane.

Our technique is as follows. We have secured for the operating room a portable x-ray machine which is spark proof and which has no high tension cables which are exposed. We have a small Bucky diaphragm which can be placed on the operating table and which contains a tunnel for the plate. The Bucky diaphragm is so placed upon the table that its center will roughly underlie the site of the patient's common duct. The rest of the table is built up with heavy pads so that the patient lies comfortably (Fig. 1). The operation proceeds as usual except that lap towels are held in place by suture and that all subcutaneous bleeders are immediately ligated, thus doing away with towel clamps and hemostats which would interfere with the roentgenogram. After the gall bladder and ducts have been exposed the common duct is carefully palpated as usual with the index finger in the foramen of Winslow. If we decide that a cholangiogram is indicated we proceed as follows:

The cystic duct is isolated as if for ligation and a ligature passed around it. A small slit is made into the duct and a ureteral catheter threaded into the cystic duct and through it into the common duct. The ligature is then tied with a single knot around the cystic duct just proximal to the opening, tightly enough to hold the catheter in place but not tightly enough to constrict it. From 5 to 20 c.c. of a sterile solution of skiodan and acacia (this is a stock preparation) are injected through the catheter into the duct.

Fig. 2.—Actual immediate cholangiogram showing three radiolucent stones. Note the cystic duct through which, in this case, the common duct was injected; the common duct is slightly dilated. Note the spill into the duodenum through the patent ampulla of Vater. For the sake of clearness the upper part of the film showing the branching of the hepatic radicals of the biliary tree is not shown.

The operative field is covered with a sterile sheet and the portable x-ray machine wheeled into place and centered.

The respiration of the patient is checked by producing a temporary apnea by means of a high oxygen concentration if the patient is having a general anesthesia, or by simply having the patient hold his breath if the operation is being done under spinal anesthesia.

The roentgenogram is taken and immediately developed in a neighboring darkroom using high-speed developers and fixatives. In less than three minutes the film is ready for viewing. Very frequently a second roentgenogram is taken to see how much of the injected media has escaped into the duodenum.

Fig. 3.—Delayed cholangiogram taken through T tube to show the ramifications of the biliary tree and the normal size relationship of the common duct.

The interval between exposures is used for general intraperitoneal exploration, removal of the appendix, when this is to be done, isolation of the cystic artery, etc. If the cystic duct is obstructed, as of course not infrequently happens, the skiodan and acacia are injected directly into the common duct with a syringe and fairly short pointed needle. Unless the common duct is very thin because of great distension the contrast media will not leak back through the puncture hole. If the common duct is greatly distended we frequently first aspirate much of the contained bile and then inject. The puncture wound can be clamped if it should leak.

The reading of the plates is an art and we realize that we are far from proficient. We realize, however, that as time goes on and we gain more and more experience the interpretation of the plates becomes easier. Frank normals are unmistakable, as are those in which the stones show

clearly. Air bubbles can be fairly easily differentiated from stones. At first we purposely injected small amounts of air during the second exposure to see whether or not air bubbles might be confusing. Edema near or at the ampulla of Vater is occasionally difficult to distinguish from actual stenosis.

In the case of multiple stones it is a great comfort to the surgeon when he finds that the number he has removed from the duct tallies with the number on the plate.

We have had no ill results from this procedure nor see why there should be any.

We have in the last two years had one experience of finding a stone which otherwise would have been overlooked. This was a patient in whom three stones, two large and one small, were seen on the cholangiogram and only the two larger ones could be palpated and were removed. Because of the third negative shadow on the film we persisted in our search and finally flushed out the smaller stone (Fig. 2). We have in several instances felt confident in the presence of a normal cholangiogram that we were correct in our decision not to open the common duct where otherwise we might have been skeptical (Fig. 3). The prolongation of the operation has been slight and we think absolutely justifiable.

After having availed ourselves of the procedure of operative cholangiography we find it difficult to understand why it is so infrequently used. We heartily agree with these authors who advocate it and we predict that within a few years it will be universally adopted.

THE EFFECT OF LIGATION OF ARTERIES OF THE STOMACH UPON ACID GASTRIC SECRETION AND UPON THE ENDOSCOPIC APPEARANCE OF THE GASTRIC MUCOSA IN THE DOG*

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AMONG the factors acting in the production of peptic ulcer, the so-called "circulatory factor" has received considerable attention. It has been demonstrated that acute experimental ulcers may be produced by causing embolic or thrombotic occlusion of the small gastric vessels. In rabbits and dogs, however, Graulich[2] and others have found that simple ligation of the *large* vessels fails to produce ulceration. This observation is particularly important to the gastric surgeon who must interfere with the blood supply to the stomach in the course of operative procedures. In an attempt to secure further information on this subject we have carried out a series of experiments in dogs to study the effect of ligation of arteries of the stomach upon acid gastric secretion and upon the endoscopic appearance of the gastric mucosa.

METHOD OF EXPERIMENTATION

Healthy adult dogs were used in this study. They had been maintained on the standard laboratory diet for four weeks before the experiments were undertaken. During this period three control measurements were made of the acid gastric secretory response to the subcutaneous injection of 1.5 mg. histamine phosphate, and gastroscopic examinations were performed to ascertain that the mucosa appeared normal.‡

Various arteries of the stomach then were divided between double ligatures and the effects of the operations upon acid gastric secretion and upon the endoscopic appearance of the gastric mucosa were studied (Tables I and II). All operative procedures were carried out under pentobarbital-sodium anesthesia and with aseptic technique. In all of the animals the position of the pylorus was changed according to Layne's technique[3] in order to permit satisfactory gastroscopic visualization of the stomach.

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‡The technique for gastroscopic examination of the dog has been described previously.[3]

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In eight dogs various combinations of arteries to the stomach were interrupted (see Table I and Fig. 1), avoiding injury to the accompanying veins. In a ninth animal the greater curvature of the stomach was devascularized completely and the spleen was removed. The animals recovered promptly and were observed for periods of from 24 to 229 days after operation. Gastroscopic examinations were performed immediately after operation and then at weekly intervals. Acid gastric secretion in response to histamine was measured at frequent intervals (see Table I).

Three of the animals in the first series (Dogs 3, 4, and 6) were used later in further experiments and all of the remaining large arteries to the stomach were ligated (see Table II). In order to effect a more nearly complete devascularization of the stomach, the splenic vessels were ligated and the spleen was removed. In Dog 3, the hepatic artery was ligated at the final operation, but in Dogs 4 and 6 the hepatic artery was not injured. In three additional dogs (10 to 12) all of the branches of the celiac axis except the hepatic artery were divided between ligatures and the spleen was removed (Table II). Gastroscopic examinations were performed before and immediately following the operation. Acid secretion was normal in these animals prior to the vascular ligation, but was not studied after operation because the survival period was short.

All animals were examined after completing the experiments to ascertain that interruption of the vessels had been effected.

OBSERVATIONS

Ligation of two or three of the large arteries to the stomach produced no significant alteration of the acid gastric secretion (Table I). Neither did it change the appearance of the mucosa as viewed through the gastroscope, except for a transient hyperemia observed immediately following operation in several animals. Such hyperemia may be observed following simple manipulation of the stomach (Layne[3]). Gastric mucosal erosions, hemorrhages, or ulcerations were not observed in any case. Even complete devascularization of the greater curvature (Dog 9) failed to change the acid gastric secretion or the endoscopic appearance of the mucosa. Microscopic sections of the gastric wall were prepared in three cases (Dogs 5, 8, and 9, which were sacrificed after 154, 58, and 58 days, respectively), and these showed no pathologic change resulting from the experimental procedures.

Ligation of all four of the main arteries to the stomach resulted in death of the animals (Table II). Death occurred whether the arterial occlusion was carried out in one (Dogs 10, 11, and 12) or two stages (Dogs 3a, 4a, and 6a). Two of the animals (Dogs 6a and 12) survived for less than ten hours. They showed many mucosal hemorrhages but

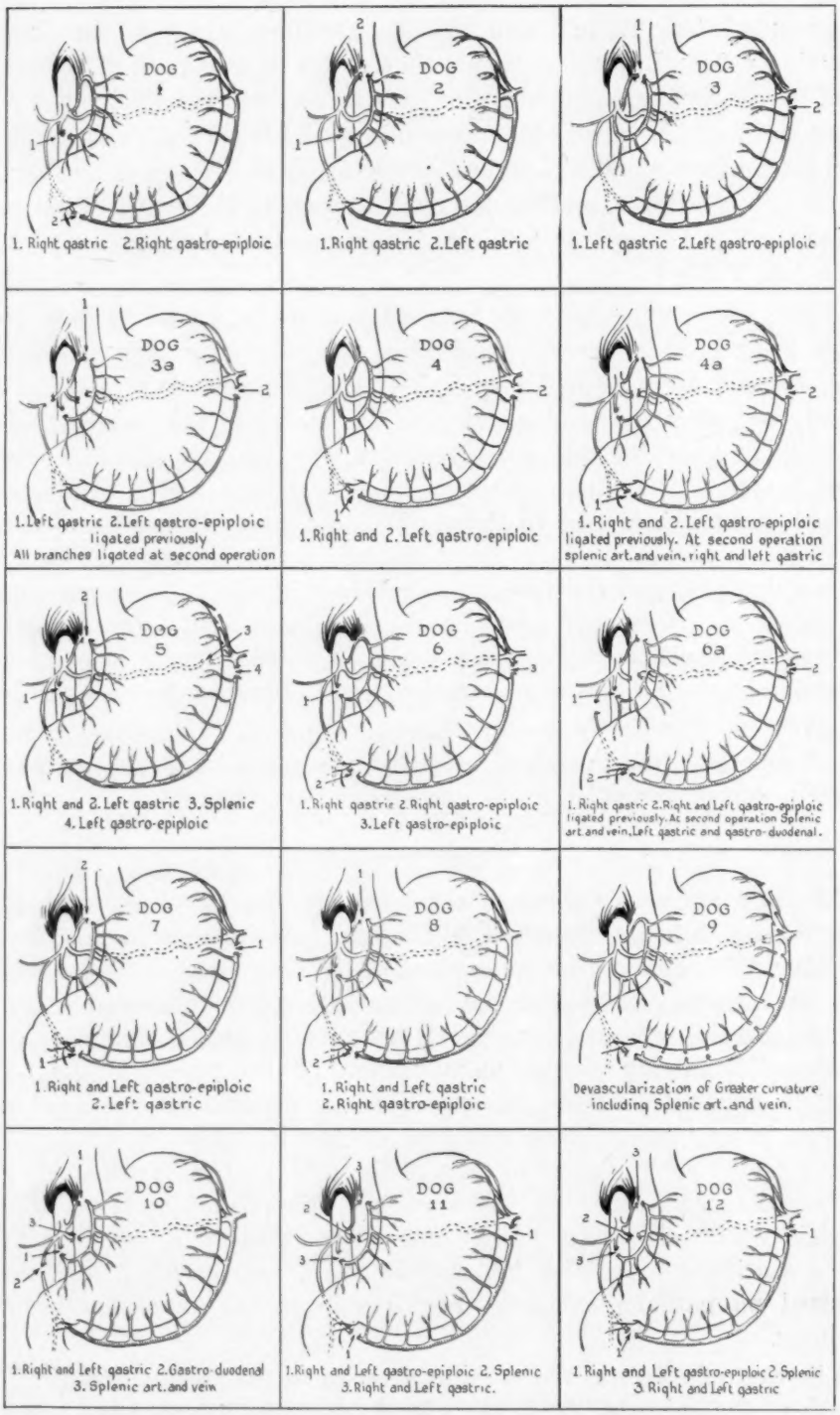
DOG 1
1. Right gastric 2. Right gastro-epiploic
DOG 2
1. Right gastric 2. Left gastric
DOG 3
1. Left gastric 2. Left gastro-epiploic
DOG 3a
1. Left gastric 2. Left gastro-epiploic ligated previously
All branches ligated at second operation
DOG 4
1. Right and 2. Left gastro-epiploic
DOG 4a
1. Right and 2. Left gastro-epiploic ligated previously. At second operation splenic art. and vein, right and left gastric
DOG 5
1. Right and 2. Left gastric 3. Splenic
4. Left gastro-epiploic
DOG 6
1. Right gastric 2. Right gastro-epiploic
3. Left gastro-epiploic
DOG 6a
1. Right gastric 2. Right and Left gastro-epiploic ligated previously. At second operation Splenic art. and vein. Left gastric and gastro-duodenal.
DOG 7
1. Right and Left gastro-epiploic
2. Left gastric
DOG 8
1. Right and Left gastric
2. Right gastro-epiploic
DOG 9
Devascularization of Greater curvature including Splenic art. and vein.
DOG 10
1. Right and Left gastric 2. Gastro-duodenal
3. Splenic art. and vein
DOG 11
1. Right and Left gastro-epiploic 2. Splenic
3. Right and Left gastric.
DOG 12
1. Right and Left gastro-epiploic 2. Splenic
3. Right and Left gastric

Fig. 1.—Schematic representation showing the arteries of the stomach which were ligated in the twelve dogs used in this experiment. The numbers identifying the animals correspond with those in Tables I and II.

TABLE I

THE EFFECT OF DOUBLE LIGATION AND DIVISION OF TWO OR THREE ARTERIES OF THE STOMACH UPON ACID GASTRIC SECRETION AND UPON THE ENDOSCOPIC APPEARANCE OF THE GASTRIC MUCOSA OF THE DOG

DOG NO.	NUMBER OF ARTERIES LIGATED	ARTERIES LIGATED	NUMBER OF DAYS OBSERVED AFTER LIGATION	EFFECT OF LIGATION OF GASTRIC ARTERIES UPON ACID GASTRIC SECRETION						EFFECT OF ARTERIAL LIGATION UPON APPEARANCE OF GASTRIC MUCOSA
				FREE ACID			TOTAL ACIDS			
				20 MIN.	40 MIN.	60 MIN.	20 MIN.	40 MIN.	60 MIN.	
1	2	Right gastric, right gastro-epiploic	Control period	36.0	65.0	68.5	45.0	69.9	72.0	No change
				57.8	69.0	62.0	62.9	74.0	64.5	
				48.9	56.0	–	53.0	61.0	–	
			14	53.4	70.6	–	62.8	79.9	–	
			24	51.2	60.7	63.0	59.9	70.0	72.1	
2	2	Right gastric, left gastric	Control period	60.8	59.0	9.0	68.0	64.1	11.7	No change
				61.2	69.0	26.9	66.6	78.0	34.4	
				48.3	34.5	–	54.1	40.0	–	
			2	50.1	43.5	58.4	59.5	56.5	69.9	
			5	21.3	58.0	45.3	30.7	64.4	51.9	
			12	55.7	61.4	61.8	67.0	72.3	76.5	
			23	52.0	78.5	72.9	56.9	82.5	77.0	
			29	44.4	58.9	28.4	50.9	63.5	33.2	
3	2	Left gastric, left gastro-epiploic	Control period	43.5	53.1	23.9	52.2	61.4	28.4	No change
				29.2	54.0	46.0	39.0	62.3	50.2	
				33.7	28.5	–	41.6	34.5	–	
				21.4	40.7	29.2	28.8	48.4	38.9	
			4	54.0	96.5	77.0	63.0	105.6	84.3	
			25	46.2	57.0	49.0	52.5	64.7	55.5	
			32	37.4	10.8	–	46.2	17.8	–	
			46	64.5	57.2	–	71.8	65.1	–	
			53	45.2	43.0	–	50.0	49.6	–	
			74	78.0	41.5	33.0	83.5	48.5	39.3	
			165	42.5	23.0	–	47.8	27.3	–	

TABLE I—CONT'D

DOG NO.	NUMBER OF ARTERIES LIGATED	ARTERIES LIGATED	NUMBER OF DAYS OBSERVED AFTER LIGATION	EFFECT OF LIGATION OF GASTRIC ARTERIES UPON ACID GASTRIC SECRETION						EFFECT OF ARTERIAL LIGATION UPON APPEARANCE OF GASTRIC MUCOSA
				FREE ACID			TOTAL ACIDS			
				20 MIN.	40 MIN.	60 MIN.	20 MIN.	40 MIN.	60 MIN.	
4	2	Right gastro-epiploic, left gastro-epiploic	Control	39.6	73.5	–	46.1	80.2	–	No change
			period	86.0	73.4	44.8	93.4	77.6	50.8	
				70.5	76.0	83.0	77.2	84.2	90.8	
				62.3	70.1	55.5	69.7	79.0	63.1	
				55.2	53.1	40.7	62.0	59.9	49.2	
				47.9	60.1	–	55.7	68.2	–	
				61.0	47.5	–	68.2	54.3	–	
5	3	Left gastric, left gastro-epiploic, right gastric, splenic artery and vein	Control	23.0	64.2	62.3	28.0	70.8	69.2	No change
			period	42.3	47.6	41.7	50.0	55.2	48.9	
				43.3	51.7	–	52.0	58.9	–	
			7	54.2	49.1	39.7	63.0	57.1	47.9	
			18	40.9	25.4	22.5	45.2	28.8	29.0	
			28	38.9	44.2	–	47.0	52.9	–	
			150	47.2	44.4	–	55.0	51.9	–	
6	3	Right gastric, right gastro-epiploic, left gastro-epiploic	Control	27.4	52.5	33.1	30.0	56.0	38.2	No change
			period	22.6	59.6	50.2	29.0	67.8	61.0	
				29.4	46.2	37.2	35.0	53.4	44.6	
			3	76.7	22.0	8.0	82.6	32.0	10.5	
			7	47.2	17.0	41.0	53.0	28.2	46.0	
			12	61.5	70.5	32.5	66.0	73.4	39.2	
			28	15.5	23.2	–	27.5	30.3	–	
			40	28.5	24.0	12.6	31.8	29.1	20.8	
			45	38.5	56.6	7.6	41.8	60.0	10.0	
			70	20.0	15.0	10.5	27.0	23.5	16.5	
			77	28.9	6.5	–	33.4	10.5	–	
			98	43.5	68.5	69.8	48.4	75.5	76.5	
			110	55.5	73.0	68.0	63.0	82.0	75.7	
			126	37.5	45.5	40.2	41.0	49.0	48.0	
			182	40.0	40.8	–	48.0	48.6	–	
			229	28.9	38.3	47.0	40.6	49.5	50.5	

7	3	Left gastric, left gastro-epiploic, right gastro-epiploic	Control period	34.3	59.1	49.0	39.1	69.8	59.5	No change
				19.8	50.0	–	27.5	57.4	–	
				32.6	41.2	32.0	38.0	47.5	39.5	
			14	21.1	43.6	10.0	26.0	48.8	16.0	
			21	44.6	55.9	–	52.0	63.0	–	
			35	52.4	67.5	39.0	60.0	75.4	47.0	
8	3	Left gastric, right gastric, right gastro-epiploic	Control period		68.2	80.0		76.1	96.6	No change
					112.0	120.1		124.0	132.4	
					92.1	128.7		102.0	132.1	
			8		88.3	105.0		96.7	112.2	
			15		126.0	122.2		134.0	128.1	
			25		104.0	118.1		114.4	126.0	
			34		122.0	76.1		128.1	80.3	
			49		124.5	116.0		130.0	124.7	
9	2	Devascularization of the entire greater curvature—arteries and veins; splenic artery and vein	Control period		116.6	128.0		122.0	138.2	No change
					112.0	62.0		128.4	74.7	
					98.2	18.7		112.3	32.1	
			6		94.4	74.0		108.8	85.0	
			15		120.0	100.5		126.0	110.6	
			25		120.8	10.6		126.9	25.0	
			34		118.0	80.8		122.7	92.0	
			39		116.2	20.7		122.0	28.8	

TABLE II

THE EFFECT OF DOUBLE LIGATION AND DIVISION OF ALL FOUR OF THE MAJOR ARTERIES OF THE STOMACH IN THE DOG

DOG NO.	ARTERIES LIGATED PREVIOUSLY	INTERVAL BETWEEN FIRST OPERATION AND COMPLETE VASCULAR LIGATION (DAYS)	ARTERIES LIGATED AT FINAL OPERATION	SURVIVAL TIME (HOURS) FOLLOWING COMPLETE ARTERIAL LIGATION OF THE STOMACH	GASTROSCOPIC EXAMINATION		NECROPSY FINDINGS FOLLOWING COMPLETE ARTERIAL LIGATION OF THE STOMACH
					BEFORE OPERATION	AFTER COMPLETE ARTERIAL LIGATION	
3a	Left gastric, left gastro-epiploic	174	All branches of celiac axis including hepatic; splenic artery and vein	10	Normal	Mild generalized pallor of the mucosa	A diffuse peritonitis was present, the middle two-thirds of the stomach was gangrenous, the liver was dark and congested
4a	Right gastro-epiploic, left gastro-epiploic	53	Splenic artery and vein, right and left gastric arteries; hepatic artery left intact	16	Normal	Mild generalized pallor	A generalized peritonitis was present, the middle two-thirds of the stomach was gangrenous
6a	Right gastric, right gastro-epiploic, left gastro-epiploic	246	Splenic artery and vein, left gastric artery, gastroduodenal artery; hepatic artery intact	6	Normal	Moderate pallor of all of the mucosa; several fresh mucosal hemorrhages along the middle part of the lesser curvature	The stomach was dilated, there were numerous submucosal hemorrhages along the greater curvature, there were no ulcerations or perforations of the stomach; the peritoneal surfaces were smooth
10			Left gastric, right gastric, gastroduodenal, splenic artery and vein; hepatic artery intact	36	Normal	Pallor of the mucosa in the middle two-thirds of the stomach	Generalized peritonitis, almost complete necrosis of middle three-fourths of the stomach
11			Left and right gastro-epiploic arteries, left and right gastric arteries, splenic artery and vein; hepatic artery intact	20	Normal	Not performed	Generalized peritonitis, gangrene of the middle three-fourths or more of the stomach
12			Left and right gastro-epiploic arteries, left and right gastric arteries, splenic artery and vein; hepatic artery intact	8	Normal	Mild generalized pallor	Many mucosal hemorrhages were present; there were no ulcerations or perforations of the stomach, peritoneal surfaces were smooth

no necrotic areas. The remaining four dogs survived for periods of 10, 16, 20, and 36 hours. They developed gangrene of the middle two-thirds to three-fourths of the stomach and a generalized peritonitis. Five of the six animals in the second series (Table II) were examined gastroscopically immediately after completion of the operation. In these a pallor of the mucosa was observed, most marked in the middle two-thirds of the stomach. Further gastroscopic examinations were not carried out because of the short survival time of the animals.

DISCUSSION

Litthauer[4] stated that the blood supply to one-third of the stomach could be cut off without producing deleterious effects. Graulich[2] and others also noted the absence of ulceration after ligation of various vessels supplying the stomach. Our studies confirmed this observation, and demonstrated that the gastric mucosa appeared normal when two or three of the large arteries to the stomach were ligated. Schindler, Necheles, and Gold[5] stated that ligation of various arteries supplying the stomach did not produce significant pathologic changes in acute experiments with dogs. (Their report does not state which arteries were ligated in individual experiments.) They stated further, however, that when the gastric mucosa was exposed to the action of $\frac{N}{10}$ hydrochloric acid or acid gastric juice stimulated by the administration of drugs, ulcerations and hemorrhages occurred in the devascularized portions, but not in the areas in which the blood supply had been left intact. In our animals no erosions or ulcerations were observed. Acid secretion remained approximately at preoperative levels, and no attempt was made to increase it except for the transient effect of the histamine phosphate injected for the purpose of measuring the acid secretory curve at intervals during the period of observation (Table I).

Our results also demonstrate that sudden, complete occlusion of the celiac axis or of the four large arteries to the stomach produces death of the dog. Blalock and Levy[1] have been able to produce gradual, complete occlusion of the celiac axis and superior and inferior mesenteric arteries in dogs without causing death or permanent injury. No abnormalities of the abdominal viscera were noted in their animals. The effect of the procedure on acid secretion was not studied. Neither was it studied in our animals because the survival period was too short.

SUMMARY

1. Ligation of two or three of the large arteries supplying the stomach produced no significant alteration of acid gastric secretion in the dog. Neither did it change the appearance of the gastric mucosa as viewed through the gastroscope.

2. Ligation of the four large arteries of the stomach of the dog resulted in death of the animal within six to thirty-six hours. Necrosis

of the middle two-thirds to three-fourths of the stomach and generalized peritonitis occurred in the animals surviving for ten hours or more.

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FEMORAL HERNIA

Description of a New Operative Procedure

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THE operative treatment of femoral hernia has stimulated the interest and resourcefulness of surgeons for many years. In the constant additions to the literature concerning the repair, two methods of approach have become standard, each of which has its staunch supporters.

One method entails the exposure and repair of the hernia from below Poupart's ligament, the other from above. Besides the difference in approach, the methods of repair differ in other respects and descriptions of procedures number well over 100. Unfortunately, follow-up studies rarely accompany these publications.

As in all operative procedures in which there are two fundamentally different methods, neither one is wholly ideal and each has features which commend it. The ideal would be an operation which combines the best features of the two approaches. Such a method has been suggested by some surgeons but by others it is considered either impossible or too difficult to undertake.

A thorough search of the literature fails to reveal a description of the repair of a femoral hernia which is here recorded and which, it is believed, combines the better features of the two standard types of operative procedure.

OPERATION

An incision is made directly over the lower border of Poupart's ligament starting at the pubic spine and extending two-thirds of the distance to the anterior superior iliac spine. The wound is deepened and the lower border of the external oblique fascia cleaned, after which the dissection is continued down to the saphenous opening. Here the hernia is freed of its superficial layers and the peritoneal sac opened. This sac is dissected free and pulled down to a point where a high ligation can be done. After ligation the sac is excised and the stump allowed to retract.

All fat and areolar tissue is now carefully removed until the femoral canal and ring are in full view. Dissection with gauze brings Cooper's ligament into the field as a glistening structure of great strength along the ramus of the pubis.

Poupart's ligament is now retracted toward the umbilicus with a small curved retractor which at the same time serves to depress this

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ligament. A gauze pad beneath the tip of the retractor protects the peritoneum from injury. The sutures, usually three in number, are passed through Cooper's ligament before being introduced through Poupart's. A small, full curved needle with a cutting edge has been found best for this purpose. The first suture is taken through the outer edge of Cooper's ligament very near the pubis and should include Gimbernat's ligament. A deep bite brings the needle out on the inner side of the ligament where it is pulled through and left on the suture. (See Fig. 1, *1*.) If the surgeon encounters difficulty in placing this

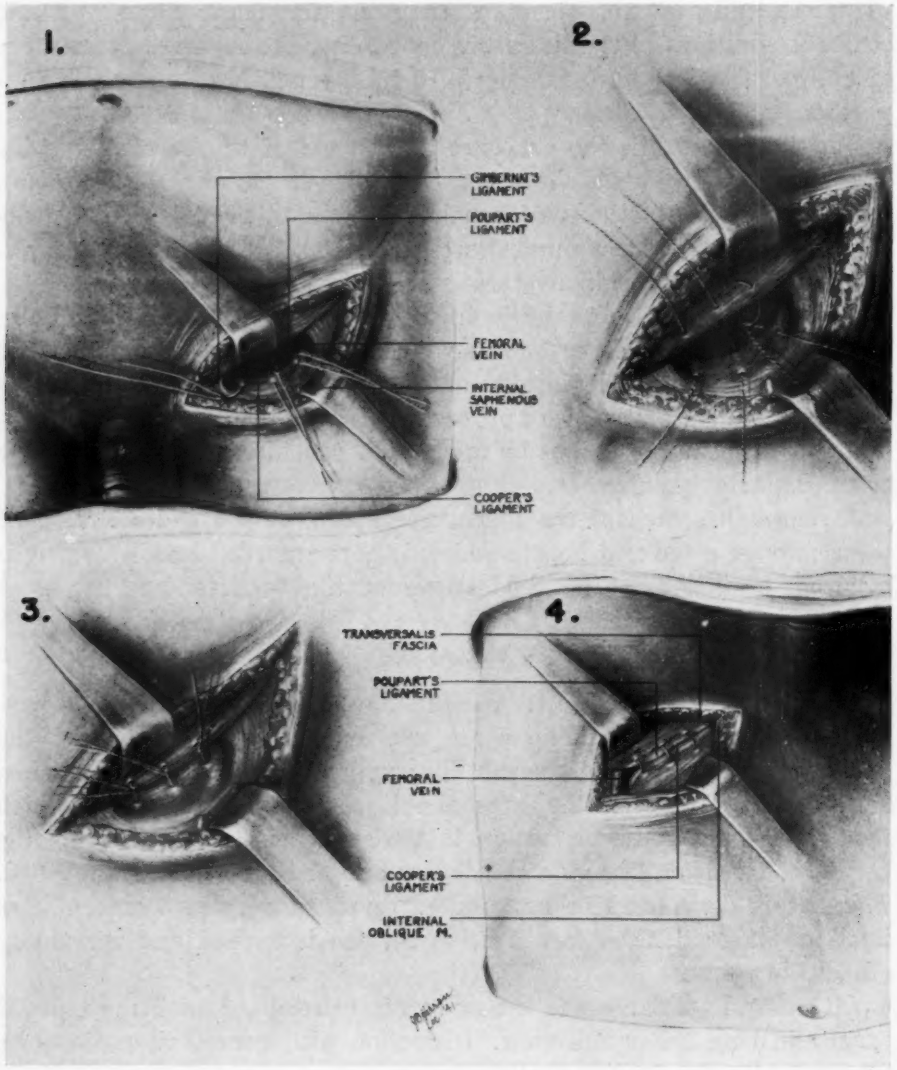

Fig. 1.—*1*, The sutures have been placed through Cooper's ligament: Poupart's ligament is depressed and retracted with a small curved retractor. *2*, The sutures are passed through Poupart's ligament, taking a broad, shallow bite. *3*, Upon tying the sutures the femoral ring is completely obliterated. *4*, An incision has been made through the entire abdominal wall to permit inspection of the repair of the femoral hernia from above; looking at it from this aspect, it is seen to have closed the femoral ring as snugly as though the sutures had been placed from above Poupart's ligament.

suture he may find it easier to accomplish from the far side of the operating table. The last of the sutures should be close to the femoral vein but not so near as to compress this vessel.

Each needle, still threaded on the suture through Cooper's ligament, is now passed through Poupart's, but this time from inside outward. (See Fig. 1, *2*.) In this instance a broad shallow bite is taken of the lower magin of the ligament. If a deep bite be taken there is danger in the male of including the spermatic cord in the suture.

Starting with the medial suture each is tied separately, bringing Poupart's into contact with Cooper's ligament and snugly closing the femoral ring. (See Fig. 1, *3*.)

Additional sutures of finer material are used to unite the lowermost border of the external oblique to the pectineal fascia and thus reinforce the repair by closing both openings of the femoral canal.

The superficial fascia is closed, the skin approximated, and a light dressing applied. After remaining in bed for a period of two weeks, the patient is allowed up progressively.

Silk is preferred as suture material for this repair.

Personal Experience.—This method of repair of a femoral hernia has been used upon numerous cadavers without difficulty and, when inspected by way of the inguinal canal (see Fig. 1, *4*), is seen to have closed the ring as completely as when repaired from above by the standard method. The operation also has been performed on both male and female patients without difficulty. Although end results on the patients are not yet available, the immediate results are most gratifying.

Anatomy.—A knowledge of the anatomy of the femoral canal and ring is most important in repairing a hernia in this region. Cooper,[1] in 1804, did much to clarify the understanding of the anatomy by publishing his studies and first described the ligament which bears his name.

On the medial side the femoral ring is bounded by Gimbernat's ligament; on the outer side lies the femoral vein; the anterior portion of the ring is formed by Poupart's ligament, and the posterior aspect or floor is formed by Cooper's ligament, which covers the ramus of the pubis. This ligament gives rise to the pectineal fascia and muscle.

PREVIOUS OPERATIONS FOR FEMORAL HERNIA

Bassini,[2] in 1894, wrote what still remains the outstanding paper on femoral hernia. In it he describes his operation for repair, carried out below Poupart's ligament. After ligating the sac and freeing the surrounding structures of all loose areolar tissue, he used three or four interrupted sutures to unite Poupart's ligament to the pectineal fascia. Several additional sutures then closed the falciform process of the fascia lata. He operated in this manner 54 times upon 51 patients,

the hernia being bilateral in three. The course of all patients was without fever or complication. All cases were followed after operation and all were without recurrence; 29 cases for from 3 to 9 years; 14 from 2 to 3 years; 5 from 12 to 18 months, and 4 less than 6 months after operation. Bassini's record has yet to be equalled.

Annandale,[3] in 1876, employed the inguinal approach for the repair of femoral hernia; the only case reported had a recurrence. In 1907, Moschkowitz[4] described the operation of uniting Poupart's to Cooper's ligament carried out through an inguinal approach and reported eighteen cases repaired in this manner without a recurrence. Ferguson[5] described his operation for femoral hernia in 1895, one year after Bassini's publication; the two procedures are very similar. Cushing,[6] in 1888, reported one case closed from below Poupart's ligament by means of a single "quilted suture," uniting Poupart's to the pectineal fascia. Other authors who have contributed to the literature, describing modifications of the two standard operations, are Lotheissen,[7] Dickson,[8] and Andrews.[9]

Follow-up Reports.—McClure and Fallis[10] report 90 cases of repair of femoral hernia. Their study shows a recurrence rate of 9.7 per cent in patients whose hernia was repaired by the femoral route and 7.4 per cent for those repaired by the inguinal route. Shelley,[11] in 222 femoral hernias repaired from below Poupart's ligament, found a recurrence rate of 3.6 per cent. Watson[12] reports 19 recurrences in 469 operations by the femoral route (4.05 per cent) and 10 recurrences in 206 operations by the inguinal route (4.85 per cent).

New York Hospital Experience.—At the New York Hospital from September, 1932, through November, 1940, a repair of femoral hernia was carried out 121 times upon 113 patients. An interval of at least six months since the last of these operations permits a review of end results. Follow-up since operation has been impossible in only two cases. The remainder have all been seen personally in the surgical follow-up clinic and examined there by a member of the surgical department.

Of the 113 patients, 95 were women and 18 men. Of the hernias, 100 occurred in female and 21 in male patients. Hernias presented on the right side in 84 instances and on the left in 37. In 5 patients the hernia was bilateral.

The ages of patients ranged from 15 to 79 years of age in the female and from 10 to 66 in the male. The duration of the hernia preceding operation varied from 1 day to 32 years.

At the time of the first operation in this hospital, 4 hernias were recurrent while five, recurring after operation here, were again subjected to operative treatment. Therefore, 9 recurrent hernias are included in this report. To date, only 1 of this group has recurred.

In 10 cases the hernia was strangulated but in only 1 was a resection of the bowel necessary.

At the time of operation associated conditions included varicose veins in 6 and diabetes mellitus in 2, while hypertension, syphilis, goiter, pernicious anemia, chronic bronchitis, and subacute bacterial endocarditis were present each in 1 case.

During this period in the New York Hospital there have been 11.4 inguinal hernias repaired to each femoral hernia.

Infection in the wounds in three patients as a postoperative complication is recorded and 1 each had a hematoma, thrombophlebitis, cerebral embolism, and cystitis In the patient who developed cystitis the bladder was opened accidentally at the time of operation.

There were no deaths after operation in the series of 113 patients.

In four patients the last visit was less than 6 months after operation; three of these died within 6 months of their discharge. Therefore, there are 115 operations upon patients who have been traced, seen, and examined 6 months to 8 years after operation. One hundred and six operations have been checked one year after they were performed. It is generally agreed that 90 per cent of hernias which recur do so within the first year after operation save when a recurrent hernia is repaired or when the wound becomes infected. In this series all recurrences were noted within a year save two, one of which had been repaired previously and one in which a wound infection developed.

In the method of repair, there have been minor variations. All operations have been divided into two groups: in the first the approach was made below Poupart's ligament (40 cases), and in the second the approach was above this structure (81 cases).

There were 8 recurrences in the 121 hernias and each of these had been repaired from above Poupart's ligament, a recurrence rate of 6.6 per cent for all cases and 10 per cent for those repaired by the inguinal route. A summary of the histories of the patients with recurrences of hernia following operation is presented later.

RECURRENCES

1 (Case 10).—A 52-year-old man had a right femoral hernia which was repaired with silk under local anesthesia. The approach was from above Poupart's ligament. After operation his convalescence was uneventful. Within one year the hernia had recurred but the patient refused another operation.

2 (Case 16).—A 53-year-old woman had a right incarcerated femoral hernia repaired with silk and under local anesthesia, the approach being above Poupart's ligament. The patient was obese and had diabetes. Her postoperative convalescence was uneventful but the hernia recurred in six months.

3 (Case 20).—A 29-year-old woman had a right femoral hernia repaired with silk and under local anesthesia, from above Poupart's ligament. Convalescence was normal but within 6 months this hernia recurred. After another operation she had remained well for 3 years. At the time of the first operation she was 4 months pregnant and was subsequently delivered of a normal child.

4 (Case 62).—A 34-year-old white woman had a right femoral hernia repaired with silk through an inguinal approach under local anesthesia. Despite a normal recovery after operation, this hernia recurred in 8 months. The hernia was again repaired and the patient has remained well for 1 year.

5 (Case 67).—A 55-year-old white woman was admitted with bilateral femoral hernia. Ten years previously, at another hospital, the left side had been repaired but the hernia recurred within 6 months. Under ethylene anesthesia a bilateral repair was carried out with catgut as suture material. On the left side, although approached from above Poupart's ligament, it was necessary to divide this structure in order to reduce the hernia. After 5 years there is a recurrence on this side.

6 (Case 80).—A 48-year-old white man, with a right femoral hernia repaired with silk under local anesthesia, developed an infection of his wound. Again an inguinal approach had been used. Three years later this hernia recurred.

7 (Case 90).—In a 51-year-old-male a right femoral hernia was repaired with silk under local anesthesia; it was approached from above Poupart's ligament. Convalescence was uneventful but the hernia recurred within 10 months. After another operation he has remained cured for 2 years.

8 (Case 116).—A 32-year-old white woman with a right femoral hernia had been operated upon in another hospital, 1 year previously, for an inguinal hernia which was not found. Repair with silk under local anesthesia and inguinal approach were followed by a smooth convalescence but a recurrence was noted 2 months later. This patient was again operated upon 2 months ago and remains well to date.

In the repair of a femoral hernia no single type of operation can be used for every case. A strangulated femoral hernia, for instance, should be approached from above Poupart's ligament so that an intestinal resection can be carried out if necessary. The method here described approaches the hernia from below Poupart's ligament but the repair is carried out as advocated by those who use the inguinal approach. Poupart's is sutured to Cooper's ligament, closing the femoral canal at its internal opening without the additional strain of a repair of the floor of the inguinal canal and also without the incision of the transversalis fascia.

SUMMARY AND CONCLUSIONS

1. A procedure which combines the better features of the two standard operations for the repair of a femoral hernia is presented.

2. The usual operations for femoral hernia are described and discussed.

3. The experiences with the surgical treatment of femoral hernia at the New York Hospital are reviewed with particular emphasis on late results and recurrences.

4. No single type of operation or approach can be used advantageously for the repair of every femoral hernia. The rate of recurrence is higher in those cases which are approached from above Poupart's ligament. Cooper's ligament may be sutured to Poupart's as effectively from below as from above Poupart's ligament.

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ACUTE SUBDURAL HYDROMA SIMULATING SYNDROME OF EXTRADURAL HEMORRHAGE

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ALTHOUGH the classical signs and treatment for acute epidural hemorrhage are well known, it is not generally recognized that following head trauma an acute unilateral collection of clear fluid in the subdural space may give rise to similar symptoms and demand prompt operative drainage of the subdural space. Naffziger,[1] Cohen,[2] Dandy,[3] Love,[4] Walsh and Sheldon[5] and others have called attention to collections of clear fluid in the subdural space following head trauma resulting in symptoms of cerebral compression often simulating subdural hematoma. This accumulation of fluid may result in acute or chronic symptoms, and since it cannot be removed by spinal drainage, an opening in the dura is necessary. It is believed that the fluid enters the subdural space through a rent in the arachnoid and becomes trapped.

Case 1 illustrates how an acute collection of this clear fluid in the subdural space can simulate the classical symptoms and signs of middle meningeal extradural hemorrhage.

CASE 1.—P. S., aged 9 years, was admitted to the neurosurgical service on May 7, 1939, with a history of head injury followed by right-sided jacksonian convulsions, right hemiparesis, and cyanosis. There was no history of previous attacks. At 2:30 P.M., one hour after the injury, twitching began of the right side of the face and the right hand, the patient became unconscious, the entire right side of the body convulsed, and the child became very cyanotic. A physician found the boy in right-sided status epilepticus, very cyanotic, and noticed that the left pupil was markedly dilated. He gave pantopon, gr. 1/6, hypodermically and in fifteen minutes the attack stopped but the patient became more cyanotic and the respirations decreased to eight per minute. The boy was admitted to the hospital and examined at 5:30 P.M. He was in deep stupor. The pupils were now constricted and equal in size. The respirations were 6 and sighing in type. The pulse was difficult to feel. Carbon dioxide in oxygen, 10 per cent, was given intranasally and 3 gr. of coramine were given subcutaneously. Lumbar puncture showed a pressure of 12 mm. of mercury with slightly pink spinal fluid. Because an epidural hemorrhage was suspected only 4 c.c. were very slowly removed, leaving a final pressure of 8 mm. of mercury. In fifteen minutes the respirations rose to 12, he reacted to painful stimuli, and it was noticed that a right hemiparesis with Babinski sign was present. The patient was then given a hypertonic magnesium sulfate enema (magnesium sulfate crystals, 3 oz.; glycerin, 1 oz.; and water, 12 oz.). By 7:30 P.M. (six hours after the injury) the respirations were up to 16, the boy opened his eyes, talked, and moved the right side slightly. The left pupil was only slightly larger than the right. At 8:00 P.M. the patient became drowsy again, the left pupil

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dilated, and the respirations decreased to 14. He became difficult to arouse. A provisional diagnosis was made of slow hemorrhage from the left middle meningeal artery and the operating room prepared for exploratory trephine. Meanwhile, x-ray of the skull showed a horizontal transverse fracture across the entire frontal bone just above the sinus level. Before operation at 9:30 P.M. the respirations had receded to 12 and the temperature was 103° F. The pulse pressure was 50. I exposed the left middle meningeal artery and found it to be intact but quite large. There was no evidence of bleeding from the anterior or posterior branch. The dura, however, was tense and bulged and upon its incision there was a copious gush of clear subdural fluid. The brain was gently retracted and at least 2 oz. (60 c.c.) of the fluid quickly ran out. Within ten minutes the patient awakened on the table and responded to questioning. A drain was placed in the subdural space and the dressings were very wet for thirty-six hours, when they were changed and the drain removed. Twelve hours after the operation there was practically complete return of function of the right side and the pupils had receded to normal size. The temperature fell by lysis in three days and the patient was discharged on the tenth day postoperatively with no complaints, talking well, and with no residual paralysis. A follow-up two years later showed a bright healthy child with no recurrence of attacks since the operation.

COMMENTS

This case illustrates that the typical symptoms and signs of epidural hemorrhage can be simulated by an acute collection of clear subdural fluid. It shows the harmful effects from the use of morphine sulfate or its derivatives in the treatment of status epilepticus, and the emergency value of intranasal carbon dioxide and oxygen, coramine, spinal drainage and dehydration in acute cerebral compression, and, finally, the therapeutic value of draining the subdural space. The large amount of clear subdural fluid removed was taking up as much space and exerting as much pressure against the brain as would an equivalent volume of epidural or subdural liquid blood or clot.

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Recent Advances in Surgery

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THE THYMUS IN MYASTHENIA GRAVIS

WITH OBSERVATIONS ON THE NORMAL ANATOMY AND HISTOLOGY OF THE THYMUS

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BLALOCK, Harvey, Ford, and Lilienthal[1] have reported the effects of removing the thymus from six patients with myasthenia gravis; more recently Blalock has removed the thymus from four additional patients. The diagnosis in each case was confirmed by the response to prostigmine salts and the intra-arterial injection of prostigmine methylsulfate. Stimulated by the marked improvement in some of these cases following operation, an investigation of the pathology of the thymus in myasthenia gravis was begun. None of the ten operative specimens examined contained a tumor. In order to establish a basis for comparison 350 thymus glands were examined between August 5, 1941, and March 23, 1942. These included approximately 200 specimens removed during routine autopsies at the Johns Hopkins Hospital and 150 specimens removed from patients who died suddenly and were autopsied at the city morgue.†

EMBRYOLOGY AND HISTOLOGY

It is usually stated that during the sixth week of embryonic life the thymus arises as ventral, cylindrical outgrowths of the third pair of pharyngeal pouches. These hollow epithelial cylinders, arising from the entoderm, soon elongate, become solid, and enlarge at their free ends. Simultaneously, parathyroid III arises from the dorsal diverticulum of the third pharyngeal pouch. During this process the mesial portions of the pharyngeal pouches, lying between these structures and the pharynx, atrophy and disappear. Thus, the two lobes of the thymus, accompanied by the detached cervical sinus, and the two parathyroids become independent structures. The caudal end of the thymus enlarges. With the great vessels and the heart, the thymus and parathyroid III move caudally towards the thorax. Parathyroid III normally ceases its descent near the lower pole of the thyroid, the thymus separating from it as it progresses into the thorax. The two lobes extend over

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†Dr. W. G. VandeGrift, assistant medical examiner, made it possible for these specimens to be obtained.

the innominate vein and upper portion of the pericardium where they form the body of the gland, although they never actually fuse. After the second month of embryonic life the solid epithelial cords are transformed into a loose network of epithelial cells. These proliferate to form rounded masses which are the lobules. The lobules are never entirely separated from one another since they represent projections from a central core of thymic tissue. There is an ingrowth of blood vessels. Connective tissue from the adventitia of these blood vessels and from the capsule of the gland forms a stroma. With the appearance of blood vessels in the thymus the interstices of the network of epithelial cells become filled with the small thymic cells and the lobules are definitely divided into a cortex and medulla.[2-4]

The origin of the small thymic cells is uncertain but they are probably identical with lymphocytes. The epithelial cells in the thymus are derived from the entoderm of the third pharyngeal pouch. During the process of differentiation of the thymus into cortex and medulla, concentric masses of cells appear in the sheets of epithelial cells of the medulla. These are the Hassall corpuscles and are commonly believed to be derived from the epithelium of the medulla.[4-6] Norris,[7] however, has recently stated that the thymus has both an entodermal component derived from the third pharyngeal pouch and an ectodermal component derived from the third branchial cleft through the cervical sinus. He believes the Hassall corpuscles are derived from the ectodermal elements and not from the epithelium of the medulla, which is of entodermal origin. This view has not yet been widely accepted.

In the normal infant, at birth, the lobules of the thymus are sharply divided into a cortex and medulla. The cortex consists of closely packed masses of lymphocytes. Scattered among these lymphocytes there are a few large cells with ovoid, pale-staining nuclei, and moderate amounts of poorly outlined cytoplasm. These are indistinguishable from the similar but more numerous epithelial cells of the medulla. Occasional blood vessels are seen in the cortex. Little connective tissue stroma seems to be present.

There is a loose stroma of connective tissue in the medulla. Many epithelial cells are present and in some areas they form irregular sheets in which the individual cell outline is lost. At varying intervals in them there are the concentric masses which are Hassall corpuscles. The Hassall corpuscles undergo hyaline necrosis and often calcification. Their number, age, and size vary greatly with the individual gland. Lymphocytes are scattered through the medulla but are present in much smaller numbers than in the cortex. Other types of cells, chiefly eosinophiles, are often found in the medulla. The blood supply of each lobule passes through the medulla into the cortex, and vessels of considerable size are present in the medulla.

In the debate about the origin of the small thymic cells and the relation of the thymus to the lymphatic structures of the body, it has been

stated that the human thymus does not contain lymphoid follicles.[8-11] Lymphoid follicles with germinal centers have been described in the thymus of the cat.[12] They have also been described in the human thymus in hyperthyroidism[13] and myasthenia gravis.[14, 15] As will be shown, the presence of lymphoid follicles with germinal centers is a prominent aspect of the changes seen in several of the thymus glands examined from patients with myasthenia gravis (Fig. 10).

The presence of lymphoid follicles with germinal centers in these glands led to a careful search of normal thymus glands for such follicles. Surprisingly enough they were found in the medulla of 14 of 150 glands from people who died suddenly without previous chronic disease. The lymphoid follicles had a typical configuration with a central area of irregular, large, pale cells grouped about a small blood vessel and surrounded by a ring of small lymphocytes. Large lymphocytes were scattered through the follicles and occasional mitotic figures could be seen. The cells forming the germinal centers differed markedly from the epithelial cells of the medulla, and the follicles did not have the stroma found elsewhere in the medulla. The lymphoid follicles were not found in the cortex.

When present in normal thymus glands the lymphoid follicles were found only in small numbers. Occasional lymphoid follicles might possibly be found in most normal glands if serial sections were cut. In this series of 350 cases they were not present in markedly involuted glands or in those of the fourteen normal infants under 3 years of age examined.

ANATOMY

In the removal and dissection of the 350 thymus glands examined, the gross anatomy was studied and, except for minor details, was found to be that given in standard textbooks of anatomy.[16, 17] The thymus in the adult is a roughly triangular gland lying beneath the sternum in the anterior mediastinum and often extending into the neck. It consists usually of two lobes which may vary greatly in size or may be further subdivided. The lobes of the thymus in the normal adult are reddish yellow in color and are firmer than the surrounding yellow mediastinal fat in which they lie and from which they can be dissected easily.

Cervical extensions of the lobes of the thymus are present in the majority of cases. These extend into the neck and may reach the base of the thyroid gland. They may be partially or almost completely separated from the thoracic portions of the gland. Often only one cervical extension is present, the other lobe being entirely intrathoracic. They are frequently connected to the base of the thyroid by thin strands of fibrous tissue, the so-called thyrothymic ligament, in which tiny blood vessels run.

The thymus is separated from the sternum by a thin but definite film of loose connective tissue which must be incised to expose the gland.

Through this run small blood vessels connecting the structures in the anterior mediastinum with the internal mammary vessels. Overlying the thymus partially on each side are the pleural reflections which must be dissected laterally in order to expose the gland adequately. The thymus may extend inferiorly over the pericardium to the third, fourth, or fifth ribs. It may extend laterally to the phrenic nerve and hilum of the lung, although this is rare in the adult. The gland lies on the pericardium and great vessels to which it is attached by strands of connective tissue, apparently a continuation of the pretracheal division of the deep cervical fascia in the mediastinum. This connective tissue also attaches the lobes to one another. The thymus is in especially close contact with the left innominate vein and it may partially surround this vessel. In the neck the cervical extensions lie beneath the pretracheal division of the deep cervical fascia and the sternothyroid muscles. They overlie the trachea and large vessels in the neck (Fig. 1).

Abnormalities in the position and shape of the thymus are not uncommon.[18] Complete absence of the gland in the infant has been reported.[19] Occasionally one lobe may be quite small or may lie entirely within the neck. The cervical extensions may reach superiorly beyond the thyroid. Thymic tissue has been described far laterally in the neck.[20] Tiny aberrant nodules of thymic tissue in the neck have been reported as occurring rather frequently in children.[21] The position of these nodules may vary greatly. Usually they are found about or within the thyroid. They have been reported in the larynx[22] and in the ear.[23] The gland may lie beneath the innominate vein and one case has been seen in the present series of 350 cases in which the lobes crossed over each other, one anterior to the innominate vein, and one posterior to it.

In the specimens examined, no aberrant nodules of thymic tissue were found. However, it was impossible to expose the neck region adequately and such nodules, even if present, might easily have been missed.

The arterial supply of the thymus (Fig. 2) is derived from three chief sources: the internal mammary arteries, the inferior thyroid arteries, and the pericardiophrenic arteries. The principal portion of this arterial supply arises from the internal mammary artery near the sternoclavicular joint. In addition to observations made on autopsy specimens and at operation, two dissections in cadavers have confirmed this. A branch supplying the thymus has also been found arising from the transverse scapular artery.*

The venous return from the thymus passes into a network of veins on the posterior surface of the thymus, between the thymus and the pericardium. Here a single large vein draining each lobe is formed. This may empty directly into the left innominate vein or may join the vein from the other lobe to form a single large vessel emptying into the left innominate vein. The inferior thyroid veins and the thyroidea ima

*The dissections were carried out by Miss Browning, the medical artist responsible for the illustrations.

usually receive branches from the cervical portions of the thymus. Small veins may empty into the internal mammary veins and a branch has been found emptying into the left superior intercostal vein in one dissection.

The lymphatic drainage of the thymus has been the source of some controversy. The lymph channels are commonly said to drain into the

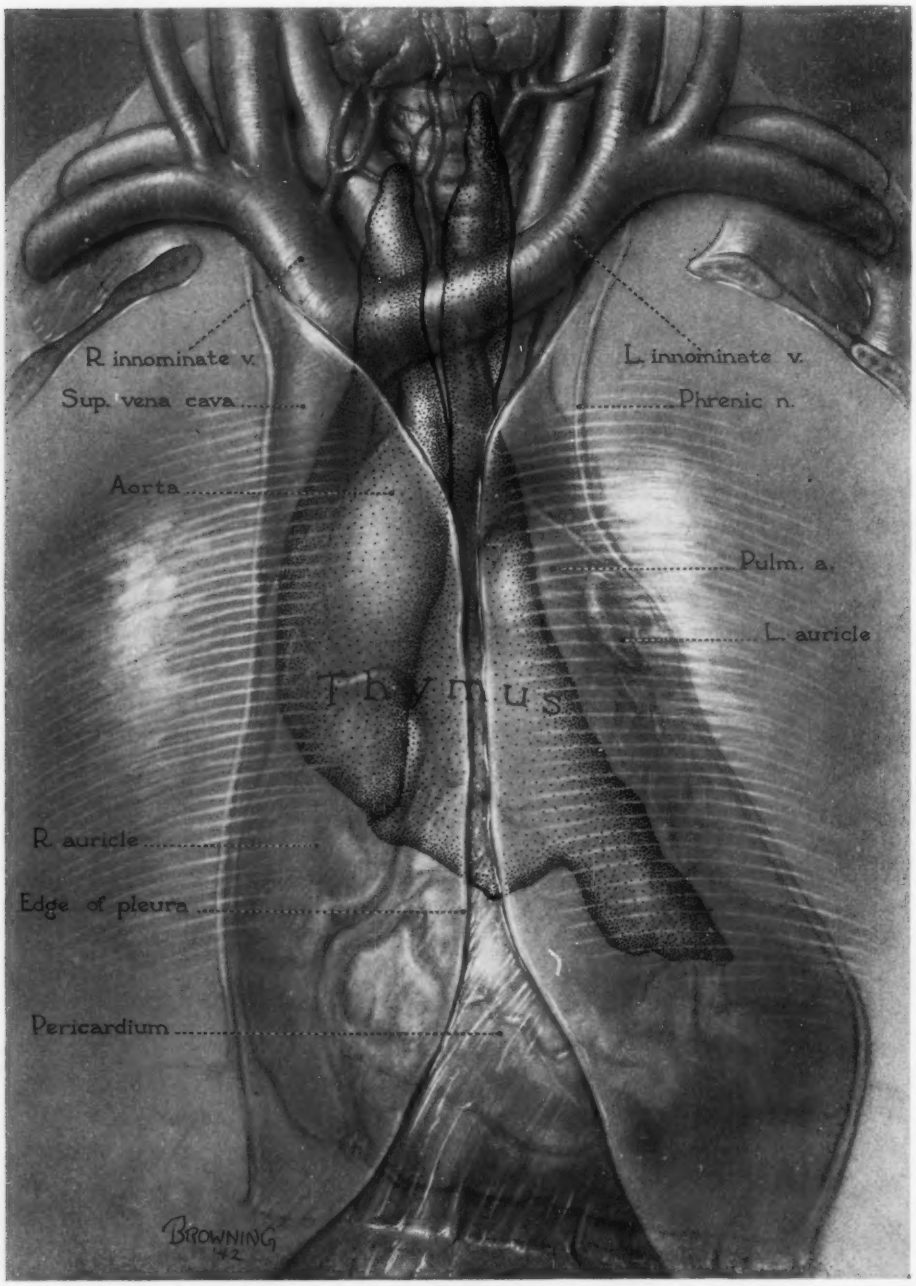

Fig. 1.—Relation of the thymus to the mediastinal structures.

superficial and deep cervical lymph nodes and lymph nodes in the mediastinum.[24] It has been stated, however, that there is a closed lymphatic system between the thyroid and the thymus, the thymus serving as a reservoir for thyroid secretions.[25] There is little to substantiate this viewpoint.[24]

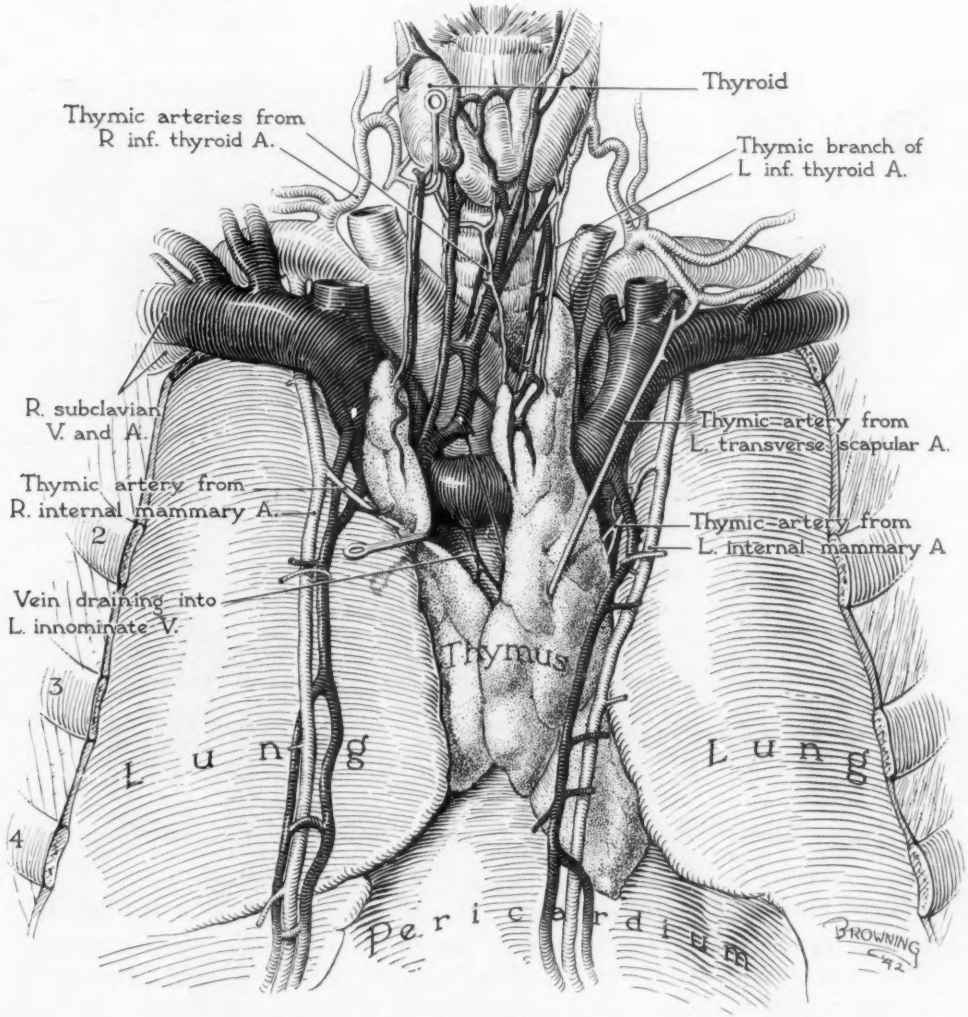

Fig. 2.—Vascular supply of the thymus.

The innervation of the thymus is similar to that of the thyroid and parathyroid glands. It arises from the cervical sympathetic chain and the vagus.[26] The nerve endings are traceable chiefly to the walls of the blood vessels.

INVOLUTION OF THE THYMUS (STUDY OF 350 CASES)

In examining the 350 thymus glands available for study from individuals dying suddenly and those dying after various illnesses, the well-known facts about the involution of the thymus were easily confirmed.[27-29] Attempts to employ the former group as a standard of

normal served to emphasize certain facts which have not been understood by previous writers describing the thymus in patients with myasthenia gravis.

In the child the thymus is a thick, solid, pinkish-gray gland which fills the anterior mediastinum. At about the age of puberty the gland undergoes marked changes. It becomes longer and more narrow as it comes to occupy a relatively smaller portion of the anterior mediastinum, although it actually may be larger than in childhood. The gland undergoes involution; that is, much of the thymic tissue present is replaced by adipose tissue lying between and widely separating the individual lobules from each other, and simultaneously the number of lymphocytes

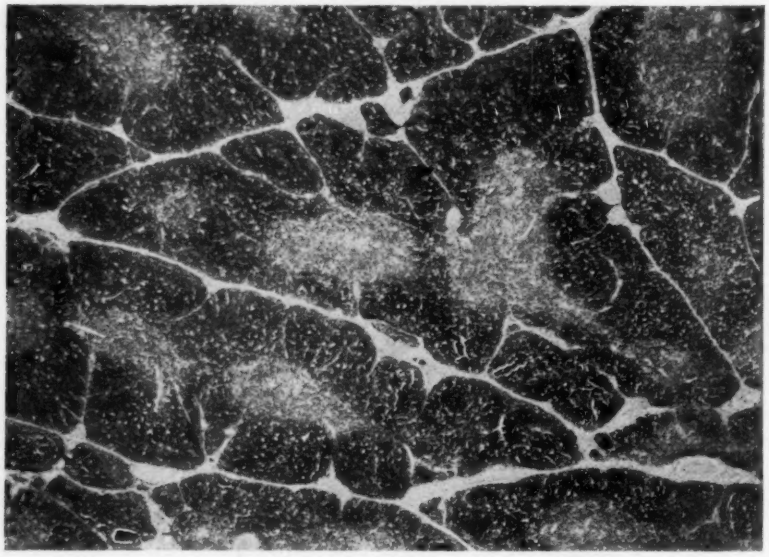

Fig. 3.—Normal thymus of a child (11-year-old colored girl). The cortex and medulla are sharply differentiated; there is no age involution and the lobules are not separated by adipose tissue (magnification ×20).

in the cortex and medulla diminishes. However, with rare exceptions thymic tissue can be found in any person, whatever his age, duration of illness, or cause of death. The thymus can still be recognized as two masses of fatty tissue which preserve the outlines of the lobes of the thymus and which can be dissected easily from the surrounding mediastinal fat. This tissue will contain small amounts of thymic tissue which can be identified easily on microscopic examination. If the patient is emaciated and has lost most of the body fat, the thymus will be present as two thin, translucent tongues of grayish tissue overlying the pericardium.

Histologically in the child the gland consists almost entirely of thymic tissue (Fig. 3). The cortex, filled with closely packed lymphocytes, surrounds and overshadows the medulla, from which it is sharply demarcated. The well-formed lobules are separated only by thin strands of connective tissue. There is no adipose tissue between the lobules.

At about the age of puberty the gland begins to undergo a process known as age involution. This atrophy or age involution consists, as mentioned above, of a decrease in the amount of thymic tissue present in each lobule and the separation of the lobules from one another by masses of adipose tissue. In the involuting thymus the lymphocytes first decrease in number and later there is a reduction in the number of epithelial cells present. It should be emphasized that in this process, although the relative amount of cortex decreases considerably, the differentiation of cortex and medulla remains distinct until the gland is so greatly involuted that only a few small, widely separated islands of thymic tissue remain in which there can be seen epithelial cells, scattered lymphocytes, and rare hyaline or calcified Hassall corpuscles (Figs. 4 and 5).

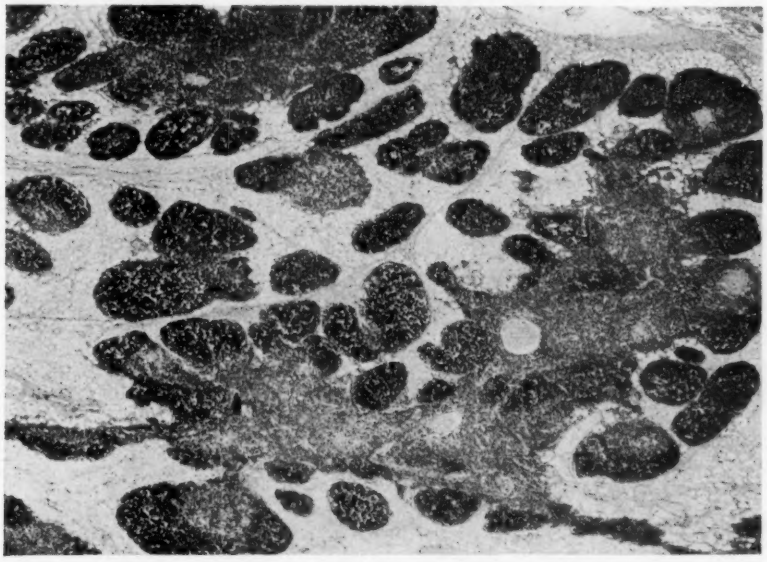

Fig. 4.—Normal thymus of an adult (a white male, 37 years of age); there is moderate age involution. The cortex and medulla are sharply demarcated (magnification ×20).

At any point in this process of age involution there may be an acceleration called accidental involution, brought on by illness[27] (Fig. 6). In a few days, even in infants, the lymphocytes may be markedly decreased in number, leaving chiefly the stroma and irregular sheets of epithelial cells. There is no evidence of continued growth of adipose tissue between the lobules during this accidentally accelerated involution, and the adipose tissue may atrophy and actually decrease in amount.[29]

The most interesting observations were made in patients meeting accidental death in the same age group as the patients in this series with myasthenia gravis, i.e., 20 to 45 years. Roughly the weights of these glands paralleled those recorded by Hammar.[30] However, just as in his cases, the extremes of weight were great, so great in fact that an average weight for a given age group was meaningless. Any gland could be

considered abnormal only if it exceeded the maximum weight recorded in the series. In addition, for any given age the thymus glands removed from normal individuals showed greatly varying degrees of involution. Some had undergone marked involution, consisting largely of fatty tissue and containing small lobules of thymic tissue. The thymic tissue present, however, showed a sharply distinguishable cortex and medulla. In

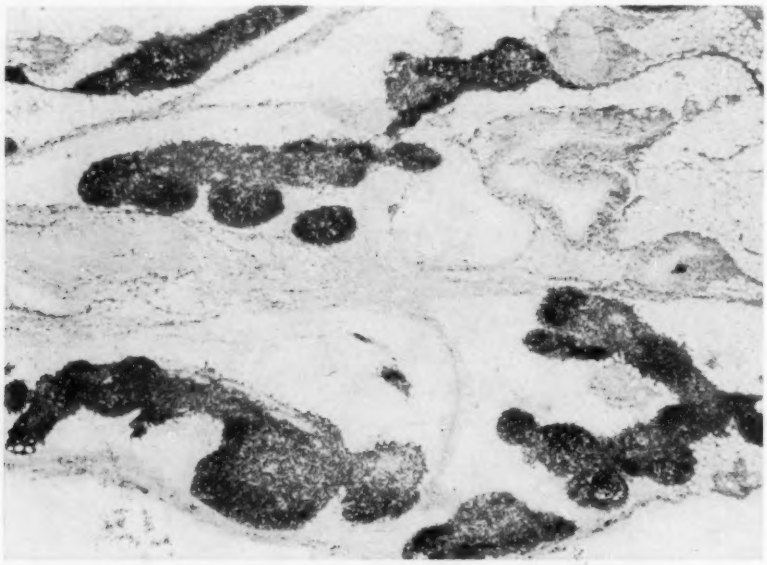

Fig. 5.—Normal thymus (magnification ×20) of an adult (39-year-old white woman). Although from an individual of approximately the same age as that in Fig. 4, there is a much greater degree of age involution.

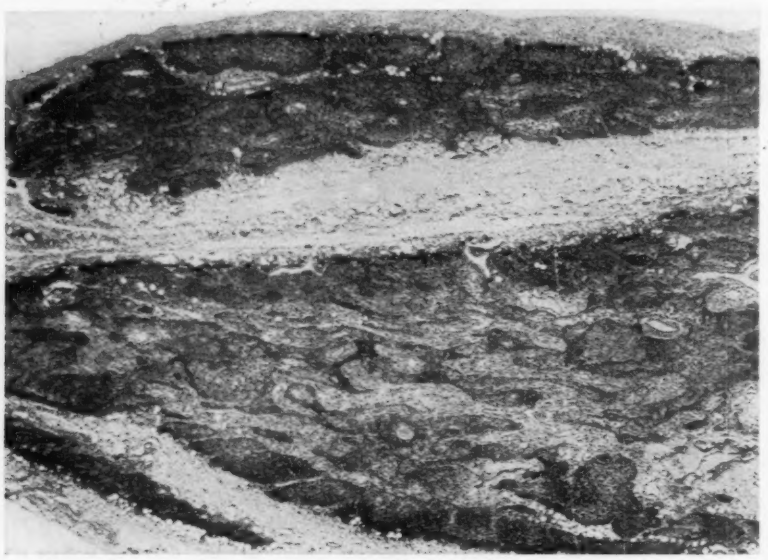

Fig. 6.—Thymus of a young adult (colored female, 19 years old) who died after a chronic illness; there is marked accidental involution. The epithelium is prominent and is as great in amount as that in the thymus glands of patients with myasthenia gravis (magnification ×20).

others there was little involution. The amount of fat present was small and the gland was made up of large lobules of thymic tissue in which the cortex and medulla were clearly demarcated, the cortex being relatively smaller in relation to the medulla than in the child (Figs. 4 and 5). This great variation in the amount of thymic and adipose tissue present in glands from normal individuals of the same age is clearly demonstrated in Hammar's diagrams.[30] Thus, several factors served to make meaningless any figures arrived at as the average normal weight of the thymus in an effort to establish significant standards by which to judge the size of the thymus glands removed from patients with myasthenia gravis. One was the great variation in the total weights of normal thymus glands. The others were the variations in the actual amounts of thymic tissue and of adipose tissue present even in glands of similar weight.

THE THYMUS IN TEN CASES OF MYASTHENIA GRAVIS

It has long been known that in certain diseases, notably hyperthyroidism, Addison's disease, acromegaly, and myasthenia gravis, the thymus may not atrophy, as in many illnesses, and that it may show certain rather characteristic changes. In some examples of myasthenia gravis the pathologic lesions found at autopsy may not be marked and may be limited to changes in the thymus. Norris[31] stated that 50 per cent of the cases of myasthenia gravis showed gross lesions of the thymus, and he believed that such lesions would be found in direct proportion to the care with which they were sought. In a series of fifty-four cases collected by Blalock and his associates[32] in which lesions of the thymus were present, these including all reported cases and one of their own, thirty-two showed tumors and twenty-two showed what was listed as persistence of hyperplasia. Lievre[33] recorded sixty-seven patients with myasthenia gravis in whom complete autopsies were performed. In twenty-four of these there was a mediastinal tumor in the region of the thymus. In thirty-two there was what was called persistence or hypertrophy of the thymus. In eleven no lesions were found. Of the five cases of myasthenia gravis autopsied at the Johns Hopkins Hospital and reported by Miller,[14] two showed tumors of the thymus, two were reported as being hyperplastic, and in one no recognizable thymic tissue was found at autopsy. In a recent case autopsied at this hospital there was a malignant tumor of the thymus with metastases to the left pleural surfaces. Of the eight cases of myasthenia gravis autopsied at the Mayo Clinic[34] one showed what was called nodular hyperplasia and the other a malignant thymic tumor with metastatic implantations on the pleura. No lesions were found in six cases. The total number of autopsied cases of myasthenia gravis reported up to the present time, including eight autopsied at the Mayo Clinic, is now stated to be ninety-nine, and in 45 per cent of these, anatomic lesions of the thymus have been found.[35]

Many of the thymus glands recorded in the literature as pathologic are listed as persistent, enlarged, or hyperplastic glands. It has been demonstrated that mere persistence of the thymus is not pathologic. The weights recorded in many of the so-called enlarged glands lie well within normal variations of weight for the thymus. Of the twenty-two glands listed in Blalock's series[32] as showing persistence or hyperplasia, the weight is recorded in ten. In only four is the weight in excess of normal and one of these cases, that of Mott and Barrada,[36] is probably a thymic tumor. The thymus glands reported as being hyperplastic have been described as showing lymphoid or epithelial hyperplasia without any detailed description of the changes present. Most attention has been centered on the tumors of the thymus in myasthenia gravis.

The material to be presented here provided an opportunity to examine ten thymus glands in which no tumor was present and to compare them with thymus glands of individuals in the same age group meeting sudden or accidental death. The clinical aspects of six of these cases have been reported by Blalock and his co-workers.[1] In the six cases when the glands were removed at operation, representative blocks were taken and the remainder quickly frozen for later extraction. For this reason the gross descriptions of these thymus glands are somewhat meager. Only one of the glands was weighed and no measurements were taken in two cases. At a later date the portions of the glands which had been frozen were thoroughly sectioned to ascertain the presence of small tumors which might have been missed previously. At this time further blocks were taken for microscopic examination. The thymus glands in the four additional cases were accurately weighed and measured, none being taken for extraction.

REPORT OF CASES

CASE 1.—P. C., a 33-year-old colored male, had an eight months' history of increasingly severe weakness, requiring before operation a total daily oral dose of 240 mg. of prostigmine bromide before he could get out of bed and feed himself. There was no clinically recognizable lymphoid hyperplasia.

At operation the thymus was found to extend in the midline from the base of the thyroid to the fifth costal cartilage. Made up of well-formed, firm tissue, the gland could not be confused with fat. (The operator identified but a single lobe of the thymus and the possibility remains that the patient retains his other lobe.) The tissue removed was not weighed or measured and no tumor was found.

Microscopically, the thymus showed evidence of involution, for the lobules of thymic tissue were separated by considerable amounts of fatty tissue. The cortex consisted of small masses of closely packed lymphocytes. It was poorly demarcated from the medulla because the medulla was diffusely infiltrated with many lymphocytes. In places the medulla contained rounded accumulations of lymphoid cells and in two areas were small groups of large, pale, irregular cells resembling those seen in germinal centers. These were not surrounded by the ring of small lymphocytes usually seen in lymphoid follicles. Considerable numbers of epithelial cells were present in the medulla. Despite the infiltration with lymphocytes, the epithelial cells in places formed irregular sheets. However, no evidence of epithelial hyperplasia was present. A moderate number of Hassall corpuscles was seen and most of these showed hyaline necrosis.

CASE 2.—R. L., a white woman, 28 years of age, had progressively severe weakness for a year. On admission she was taking 1 Gm. of guanidine and six or seven 30 mg. doses of prostigmine bromide each day. Still she was confined to bed. There was no clinical lymphoid hyperplasia.

At operation, a gland twice the size of that in Case 1 was found. It was formed of two lobes which began at the lower poles of the thyroid, extended downward as two separate lobes to the level of the third costal cartilage, and then formed a single mass which terminated at the level of the fifth costal cartilage. The thymic tissue was grossly firm and well formed. No measurements were taken. There was no tumor present.

On microscopic examination there was a moderate degree of involution, for the distinct lobules of the gland were separated from one another by adipose tissue. Again there was considerable amount of cortical tissue consisting of rather closely packed masses of lymphocytes. The cortex was greater in amount and the lymphocytes more closely packed than in thymus glands with a similar degree of involution in individuals of the same age. The differentiation between cortex and medulla was less sharp than normal. The medulla was diffusely infiltrated with lymphocytes and these tended to separate the epithelial cells so that they were scattered individually through the medulla and did not form the irregular sheets usually seen. There were no lymphoid follicles present in the medulla and there was no epithelial hyperplasia. The many large Hassall corpuscles showed hyaline necrosis.

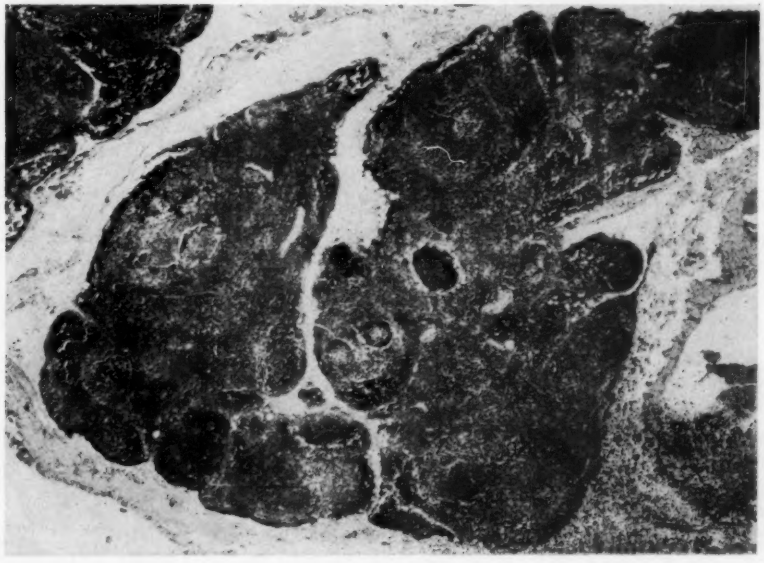

Fig. 7.—Thymus of patient in Case 3 (22-year-old colored female). There is a great increase in the number of lymphocytes present, especially in the medulla; the demarcation between cortex and medulla is indistinct. Lymphoid follicles with germinal centers can be seen (magnification ×20).

CASE 3.—L. K., 22-year-old colored woman, had a six-year history, the disease being moderately severe for three and one-half years. On admission she required 105 to 120 mg. of prostigmine bromide a day but remained severely handicapped by her illness. There was no clinical evidence of lymphoid hyperplasia.

Operation disclosed a large, firm thymus with well-defined right and left lobes. These extended from the lower poles of the thyroid to the level of the third interspace and in their thoracic positions spread rather far laterally. The right lobe measured 9 by 2.5 by 0.8 cm., and the left lobe 15 by 2.5 by 0.8 cm. No tumor was found.

Involution in this gland (Fig. 7) was slight and it consisted almost entirely of thymic tissue. Areas of cortex were seen but these were so poorly demarcated from the medulla, which was densely infiltrated with lymphocytes, that each lobule presented an almost homogeneous appearance. In the medulla there were numerous large, lymphoid follicles with prominent germinal centers. In many of these, mitotic figures were present. The epithelial cells in the medulla were widely separated and almost obscured by the many lymphocytes. There was no evidence of epithelial hyperplasia. Few Hassall corpuscles were present in the gland.

The lack of involution in this thymus was not unusual, for occasional glands in normal individuals of the same age showed as little involution as was present here. However, in such normal thymuses the cortex and medulla were sharply demarcated and each contained fewer lymphocytes.

CASE 4.—H. M., a woman 39 years of age, had a five and one-half-year history of mild myasthenia gravis, weakness being limited largely to those muscles innervated by the cranial nerves. The patient died following operation.

Operation showed a moderate amount of thymic tissue above the innominate veins, although the majority of the gland lay over the pericardium. The two large lobes extended from the midline rather far to either side, the left lobe reaching the hilum of the left lung. The right lobe measured 9 by 3 by 0.8 cm., the left lobe 15.5 by 2 by 0.5 cm. There was no tumor present.

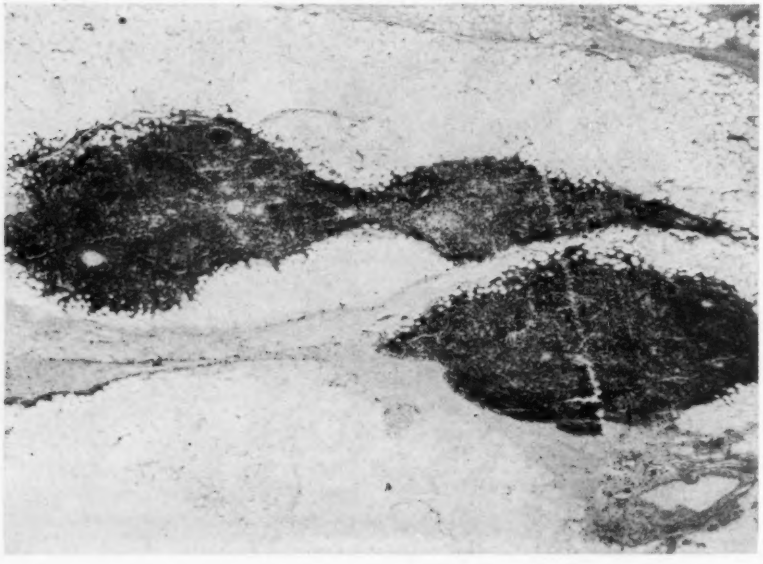

Fig. 8.—Thymus of Case 4 (a white woman, 39 years of age). Although there is marked age involution, the increase in the size of the lobules due to the increased number of lymphocytes present is easily seen (magnification ×20).

Sections of this gland (Fig. 8) differed in some respects rather markedly from those already described. Involution was far advanced, for the thymus contained large amounts of adipose tissue and only small scattered lobules of thymic tissue, but these lobules were much larger than in normal glands showing a similar degree of involution. No differentiation between cortex and medulla was visible. Instead, there was diffuse infiltration of each lobule of tissue with lymphocytes which could be seen spreading into the fatty tissue about the remnants of the thymus. Several lymphoid follicles with germinal centers were present. The epithelium was overshadowed by the lymphoid cells and only small amounts were seen. Rare, small Hassall corpuscles were found.

In normal glands at this stage of involution few lymphocytes were present and the small lobules consisted almost entirely of epithelial cells and stroma.

Autopsy was limited to the thorax and none of the endocrine glands were obtained for examination. The thymus had been completely removed at operation. There was no lymphoid hyperplasia present in the thorax.

CASE 5.—R. S., a 30-year-old white woman, had for seven months moderately progressive weakness until before operation she required 120 mg. of prostigmine bromide each day. There was no clinical lymphoid hyperplasia.

Most of the thymic tissue found at operation lay inferior to the innominate vein but there was a horn extending up on the left side to the inferior pole of the thyroid. At the upper part of the heart the lobes were broad and extended far laterally. The thymic tissue was firm. The right lobe of the thymus measured 8 by 2.5 by 0.8 cm., the left lobe measured 8 by 2.5 by 0.8 cm., while its cervical projection measured 9 by 6.1 by 0.8 cm. The gland weighed 30 gm. There was no tumor.

Microscopic examination of this gland showed considerable involution, for the lobules of thymic tissue were rather widely separated by fatty tissue. The changes in this thymus were less marked than in any other of the series. The cortex and medulla were rather sharply differentiated. Epithelial cells were easily distinguishable in the medulla and numerous Hassall corpuscles showing varying degrees of hyaline necrosis were present. There was no epithelial hyperplasia; however, the cortex showed a greater number of more closely packed lymphocytes than normal. There were abnormal numbers of lymphocytes in the medulla, and in places they formed small rounded accumulations. No lymphoid follicles with germinal centers were found.

CASE 6.—M. W., a white woman, 34 years of age, had a two-year history of progressive weakness of moderate severity. On admission, 15 mg. of prostigmine bromide every two hours both day and night did not permit her to walk by herself. There was no clinical lymphoid hyperplasia.

At operation the bilobed thymus extended from the thyroid gland to the fourth costal cartilage or slightly lower. Each lobe measured approximately 12 by 2.5 by 0.8 cm. There was no tumor.

The picture seen on microscopic examination of this thymus was like that seen in several of the other glands. There was a moderate degree of involution. The cortex was densely packed with lymphocytes and was more abundant than in normal glands showing a similar degree of involution. The medulla was heavily infiltrated with lymphocytes and the demarcation between cortex and medulla was indistinct. Numerous large lymphoid follicles with germinal centers were present in the medulla. The epithelial cells were partially obscured by the many lymphocytes about them. There was no epithelial hyperplasia. Many Hassall corpuscles showing hyaline necrosis were present.

CASE 7.—L. G., a white woman, 23 years old, had a five-year history of progressive weakness with remissions during two pregnancies. During the eight months preceding operation the patient's symptoms were well controlled by 150 mg. of prostigmine each day. There was no clinical evidence of general lymphoid hyperplasia.

At operation a firm, bilobed thymus was found. The right lobe measured 16 by 3 by 0.8 cm., the left lobe 14.5 by 3 by 0.8 cm. The gland weighed 28 Gm. There were definite cervical extensions into the neck, the right lobe extending 1.5 cm. higher than the left. The thymus grossly showed evidence of involution but many pinkish-gray lobules of thymic tissue were visible on section. On careful examination there was no tumor to be found.

On microscopic examination the gland showed definite evidence of involution, for the lobules were separated by small amounts of adipose tissue. A recognizable cortex

and medulla were present but both were densely infiltrated with lymphocytes and the normal, sharp demarcation of cortex and medulla was absent. Numerous large lymphoid follicles with germinal centers were present in the medulla. In this thymus the epithelium of the medulla could be seen plainly as irregular sheets of cells but the epithelium was no more prominent than in the glands of many individuals without myasthenia. Only rare small Hassall corpuscles were present.

CASE 8.—M. C., a 37-year-old white woman, had a nine-year history of progressive weakness. For two years the patient had remained in bed, a helpless invalid, despite large amounts of prostigmine. No general glandular enlargement was present.

At operation a small thymus gland was found overlying the base of the heart and the great vessels. There were no cervical extensions. The gland was dissected free with some difficulty and was not as sharply demarcated from the surrounding mediastinal fat as in some of the other cases. The right lobe measured 10 by 1.5 by 0.7 cm., and the left lobe 9.5 by 1.5 by 0.7 cm. The gland weighed 13 Gm. The thymus was firm, large and yellow-red in color. On section, numerous large scattered lobules of thymic tissue were seen. No tumor was found.

On microscopic examination the thymus showed characteristic changes. There was moderate involution and the lobules of thymic tissue were separated by considerable amounts of adipose tissue. The individual lobules of thymic tissue were larger than those seen in normal glands showing a similar degree of involution. This increase in the size of the lobules of thymic tissue was due to an increase in the number of lymphocytes present in the cortex and medulla of each lobule. The normal sharp differentiation between the cortex and medulla was lost. In the medulla there were large numbers of lymphoid follicles with prominent germinal centers. Irregular sheets of epithelial cells were present but these were no more prominent than in the glands of many individuals without myasthenia. There were only a few small Hassall bodies.

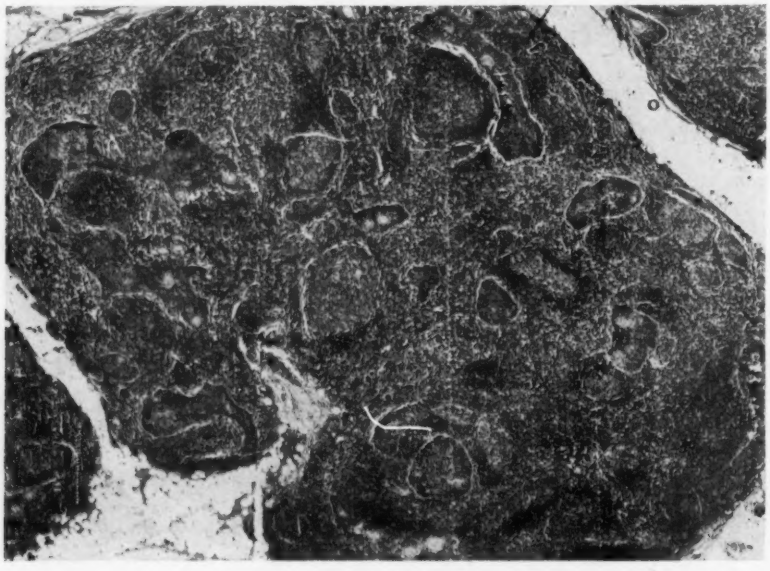

Fig. 9.—Thymus of Case 9 (a 24-year-old white woman). The increase in the size of the lobules and the large number of lymphoid follicles with germinal centers are clearly demonstrated (magnification ×20).

CASE 9.—M. W., a white woman, 24 years old, following the birth of her second child four years before admission, began to have increasing nervousness and definite exophthalmos. Basal metabolic rate was reported as plus 30 previous to a thyroidec-

tomy two years before admission. Following this the exophthalmos continued. A month after her thyroidectomy the patient had progressively increasing weakness until she remained a semi-invalid despite 45 mg. of prostigmine every four hours. There was no clinical lymphoid hyperplasia.

At operation a small, firm, well-demarcated, bilobed thymus was removed. The right lobe measured 12.5 by 3.5 by 1.0 cm., the left lobe 10.5 by 1.5 by 0.8 cm. The gland weighed 15 Gm. There was no tumor.

There was involution of the thymus on microscopic examination for the lobules were separated by adipose tissue (Fig. 9). Each lobule was larger than normal due to a marked increase in the number of lymphocytes present in both the cortex and medulla. The normal sharp demarcation between cortex and medulla was lost. In the medulla were large numbers of lymphoid follicles with prominent germinal centers. There were also in the medulla irregular sheets of large, pale cells with poorly outlined cytoplasm. These epithelial cells were present in considerable numbers but there were no more of them than can be found in many normal glands in patients of the same age group. There were moderate numbers of Hassall's corpuscles present. These varied considerably in size and most of them were hyaline.

CASE 10.—H. M., a white woman, 27 years of age, had a two-year history of progressively severe weakness. Six months after the onset of her illness she delivered a child in a normal labor. On admission the patient was taking 90 to 135 mg. of prostigmine each day. On this she did fairly well but each morning after a night without prostigmine she was unable to raise her arms. There was no clinical lymphoid hyperplasia.

At operation a well-formed thymus with two lobes extending from the inferior pole of the thyroid to the fourth interspace was found. The right lobe measured 13 by 3 by 0.8 cm., and the left lobe 15.5 by 2.2 by 0.7. The gland weighed 25 Gm. No tumor was present.

On microscopic examination the thymus showed definite involution for the lobules were separated by small amounts of adipose tissue. Each lobule was larger than normal and this increase in size was due to the presence of large numbers of lymphocytes in the cortex and medulla. The normal sharp differentiation between cortex and medulla was lost. In the medulla there were large numbers of lymphoid follicles with prominent germinal centers. The epithelial cells in the medulla formed small, irregular sheets. Only a few, tiny Hassall corpuscles were present. There was no epithelial hyperplasia.

DISCUSSION

In two of Miller's series[14] of five cases of myasthenia gravis no tumor was found and the thymus glands were said to show epithelial hyperplasia. These cases have been re-examined with the changes described in mind. One thymus in a 45-year-old white man with an eight-month history of progressive weakness weighed 60 Gm. and each lobe measured 7 by 3 by 1.5 cm. The picture presented was much like Case 4 of this series. The sections showed a considerable degree of involution, there being relatively little thymic tissue in a fatty gland. No definite distinction between the cortex and medulla was present and the tissue was uniformly infiltrated with lymphocytes. In the medulla were many large lymphoid follicles with prominent germinal centers. The amount of epithelium present was not in excess of normal and there was no evidence of epithelial hyperplasia. The Hassall corpuscles showed hyaline necrosis and calcification. No generalized lymphoid hyperplasia was found in the other organs.

The other case of myasthenia gravis in Miller's series in which no thymic tumor was found occurred in a 27-year-old white woman with a four-year history of progressively severe weakness. The thymus weighed 30 Gm. and measured 6 by 6 by 0.5 to 1.0 cm. The gland showed moderate involution. The cortex and medulla were densely packed with lymphocytes. In the medulla were definite lymphoid follicles with germinal centers. Moderate numbers of small Hassall corpuscles were present. There was no epithelial hyperplasia. Examination of the other organs showed no evidence of lymphoid hyperplasia elsewhere.

Norris[31] believed that epithelial hyperplasia was the chief pathologic change in the thymus in myasthenia gravis and interpreted the tumors found in myasthenia as extreme degrees of epithelial hyperplasia. Similarly, Miller[14] described such hyperplasia in his cases. Re-examination of the thymus glands in his cases did not show any abnormal amounts of epithelial tissue present. Instead the changes in them were similar to those in the present series.

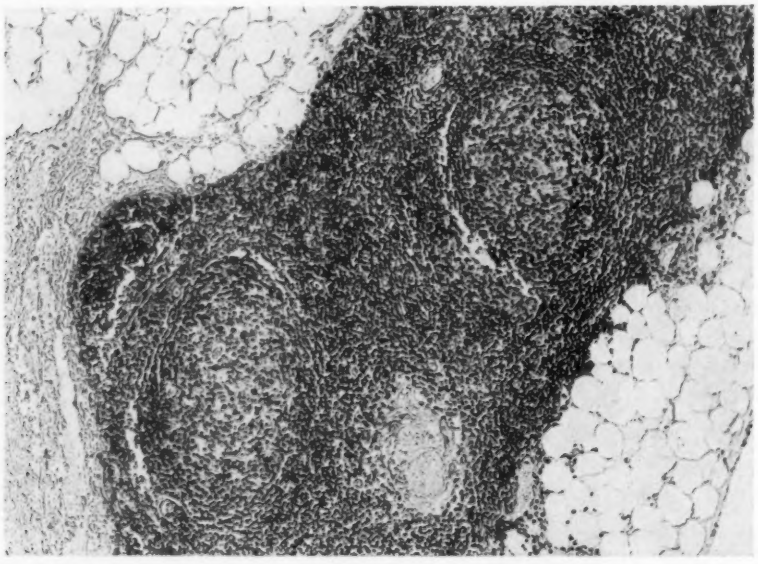

Fig. 10.—Higher magnification (×90) of Case 6 (a white woman, 34 years of age) to show lymphoid follicles with germinal centers.

In this series of ten cases, certain constant changes were present in each thymus (Figs. 7, 8 and 9). First, there was involution, although the degree of involution varied greatly. This was the so-called age involution present in all normal adult thymus glands. It consisted of a replacement of the thymic tissue by fat and a resulting separation of the lobules of the gland by adipose tissue. Secondly, the individual lobules were larger than those of the control cases, due to an increase in the numbers of lymphocytes present. In most this included a relative increase in the amount and density of the cortical tissue. In all there was an increased infiltration of the medulla by lymphocytes. In seven

of the cases this infiltration was accompanied by the presence in the medulla of abnormal numbers of lymphoid follicles with germinal centers (Fig. 10). None of the thymus glands showed evidence of epithelial hyperplasia. There were no constant changes in the character or number of the Hassall corpuscles.

It was considered possible that the changes observed in the thymus glands removed from patients with myasthenia gravis might represent part of a generalized lymphoid hyperplasia. Six autopsied cases of myasthenia gravis were examined. Five were those reported by Miller.[14] The sixth was a patient with myasthenia gravis in whom a tumor metastasizing to the left pleural cavity was found. In this case a tumor measuring 8 by 6.5 by 3 cm. was present in the anterior mediastinum in the position of the thymus. It contained both lymphoid and epithelial elements and was not different from the benign thymic tumors called thymomas. The numerous metastases on the left visceral and parietal pleuras were similar microscopically to the original tumor. There were no other metastases. Lymphorrhages were present in the muscles, but the other organs showed no lesions. No normal thymic tissue was found. In one case in which a benign tumor of the thymus was present there was slight enlargement of the mediastinal and cervical lymph nodes and the pharyngeal lymphoid tissue. In none of the others was there any evidence of generalized lymphoid hyperplasia.*

It is unfortunate that more accurate gross descriptions of the thymus glands in this series were not available. The weights of five of the glands were 30, 28, 25, 15, and 13 Gm. These lie within the range of normal. Of those glands which were measured, none was larger than some normal glands in the same age group. However, the great variation in the amounts of thymic and adipose tissue actually present in the glands makes comparisons of weight valueless. Undoubtedly a few of the thymus glands found in cases of myasthenia gravis have weighed more than normal glands. Without knowing the relative amounts of thymic and adipose tissue present, it is impossible to say whether there was actually in these cases an increase in the amount of thymic tissue. The criteria of abnormality are not to be sought in the size of the gland but rather in its microscopic appearance.

Whether or not the thymus is enlarged in myasthenia gravis when compared with normal glands may be an academic point for still another reason. Patients suffering with this disease have been subjected to a long, often debilitating illness which may, through inability to swallow, produce considerable loss of weight. Excepting the diseases previously mentioned which are also associated with abnormalities of the thymus gland, it might be expected that any such chronic illness would be associated with marked involution of the thymus (Fig. 6). Thus, in patients

*Case 4 of the present series was autopsied but examination was limited to the thorax. There was no lymphoid hyperplasia in the thorax.

with chronic illnesses of comparable duration the thymus glands are commonly much more involuted than those in patients with myasthenia gravis.

It should be emphasized that the changes found in the thymus in myasthenia gravis are not peculiar to that disease alone, for similar changes may be found in acromegaly, Addison's disease, and hyperthyroidism. It is interesting that these conditions are often associated with muscular weakness and that myasthenia gravis has been reported as occurring with hyperthyroidism.[35, 37, 38, 39]

Examination of the thymus in seven individuals with Addison's disease showed three in which changes occurred like those described in myasthenia gravis. In a fourth case there was a tumor of the thymus containing both lymphocytes and epithelial cells. Large lymphoid follicles with germinal centers were present in the tumor. Two cases occurred in children 3 and 4 years of age. The thymus glands appeared normal. In the seventh case, that of a 28-year-old white female, the thymus was markedly involuted and there was apparently no increase in the number of lymphocytes present.

The thymus was examined in five cases of acromegaly. Here the striking change was the lack of involution in all these glands. This was very much less than that found in the thymus glands of patients with myasthenia gravis. In addition, two of the glands showed the increase of lymphocytes found in myasthenia gravis.

Twenty thymus glands from individuals with hyperthyroidism were examined. In eighteen of these there was again a striking lack of involution. Five showed, in addition to a lack of involution, the changes found in myasthenia gravis (increased lymphocytes). Two glands showed a degree of involution within the limits of normal, but in these cases the epithelial hyperplasia in the thyroid was slight. Only one of the two patients received Lugol's solution.

SUMMARY

1. Examination of 350 thymus glands, of which 150 were removed from patients dying suddenly, confirmed the well-known facts about the anatomy, histology, and involution of the thymus.

2. Of the 150 thymus glands examined in patients dying suddenly, fourteen were found to contain lymphoid follicles with germinal centers in the medulla.

3. It was impossible to set up significant standards of weight for the normal thymus since great variations were found in the weight, the amount of thymic tissue, and the amount of adipose tissue present. Hence, the weight of the thymus as an index of pathologic change was considered to have little value. The ten thymus glands in this series of patients with myasthenia gravis apparently were no larger than many normal glands.

4. Examination of ten thymus glands, none of which contained a tumor, removed at operation from patients with myasthenia gravis showed constant changes.

a. In all there was some age involution. This varied from a slight degree of involution to marked separation of the thymic lobules by adipose tissue.

b. In each case there was an increase in the number of lymphocytes present in the thymus. This consisted of an infiltration of the medulla with large numbers of lymphocytes and usually a relative increase in the size and density of the cortex. The normal *sharp* differentiation between cortex and medulla was lost.

c. In seven of the cases, abnormal numbers of lymphoid follicles with germinal centers were present in the medulla.

d. There was no epithelial hyperplasia and no constant change in the character and number of the Hassall corpuscles.

5. In six autopsied cases of myasthenia gravis examined which had not undergone operation there was no generalized lymphoid hyperplasia. The changes found in the thymus in myasthenia gravis were not part of such a generalized lymphoid hyperplasia.

6. The thymus was examined in individuals with Addison's disease, acromegaly, and hyperthyroidism.

a. In seven individuals with Addison's disease, the thymus of three showed changes like those in myasthenia gravis.

b. The thymus glands in five cases of acromegaly showed a striking lack of involution. In addition, two of these glands showed the changes found in myasthenia gravis.

c. In twenty thymus glands examined from individuals with hyperthyroidism, eighteen showed a striking lack of involution. In addition, the changes found in myasthenia gravis were present in five.

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In Memoriam

CHARLES VIRGIL MOSBY
1876—1942

On November 9, 1942, Dr. Charles Virgil Mosby died in the Grace Hospital, Detroit, of heart disease. With his untimely passing there came to a close a career dedicated to the dissemination of medical knowledge and to the advancement of medical literature and education.

Dr. Mosby, the collateral kinsman of Colonel John S. Mosby, of Civil War fame, was born on August 18, 1876, in Nevada, Missouri. The youngest of six children, he grew up on a farm and early learned "the doctrine of the strenuous life" by which he lived and died. He entered the old St. Louis College of Physicians and Surgeons and was graduated in 1899, paying his way through school by selling medical books. Following his graduation, he entered medical practice and after two years gave this up to devote all his efforts to the medical publishing field. The aspiration, kindled during his medical student days, to further medical knowledge through faithful recordings of experienced observers and to enrich medical literature with stimulating thought and advances, rapidly developed into a crusading spirit. With this firm conviction, he established, in 1901, the publishing firm which bears his name and launched his first book, *The Golden Rules of Surgery,* by A. C. Bernays, well-known St. Louis surgeon. The tremendous success of this endeavor assured him of his hopes and added impetus to his desires.

Imbued with the spirit of propagating medical knowledge, Dr. Mosby traveled far and wide exhorting eminent medical educators to widen the scope of their teachings by writing textbooks which could be at the disposal of greater numbers of students and physicians. Dr. Mosby has stimulated the publication of hundreds of books, in addition to which he founded eight medical journals and one dental journal. It was Dr. Mosby's belief that "medicine and surgery would still be primitive crafts had it not been for a few dauntless souls who were willing to record faithfully by the written word the result of their findings," and with this courageous spirit he constantly extended the frontiers of American medical and dental education.

In addition to his publishing enterprise, he found time to write philosophic dissertations and biographical essays. Among the better known of these are *Making the Grade, Little Journeys to the Homes of Great Physicians, William Beaumont—Physiologist, Augustus Charles Bernays —Surgeon* and *Williams McKim Marriott—Pediatrician.* For these contributions and in appreciation of his unceasing efforts and his prolific and fructiferous activities in the publishing field, he was awarded an honorary doctor of science degree by the Bates College, Lewiston, Maine.

Dr. Mosby was one of the pioneer exponents of enhancing the qualitative contributions of carefully selected authors by presenting them in a style sufficiently attractive that they would receive the full meed of attention which they justly deserved. Dr. Mosby had the knack and appreciated the importance of presenting the printed page to the eye of the reader in such a manner that the information which the author strived to convey could be more easily comprehended with the least amount of ocular effort. He possessed the ability happily to combine literary creations which were both meritorious and at the same time pleasing to the artistic sense of the medical book lover. Because of his consistent appreciation of these all-important requisites and desiderata, his work in building epoch-making medical publications will long endure as an outstanding example of what may thus be accomplished.

All who knew Dr. Mosby, and his friends were legion, were forcefully impressed with his sterling human qualities, his indomitable resoluteness, his indefatigable energy, his untiring industry, and his idealistic attitude tempered by a realistic zeal. His clear penetrating eyes, determined chin, and pleasing countenance vividly reflected his beneficent cordiality, his contagious enthusiasm, and his innate integrity. All of these admirable characteristics were harmoniously blended to distinguish his charming and generous personality and to instill immediate and reassuring confidence. Indicative of his magnanimous character is his expressed gratification of his intense interest in his work, best exemplified by his own words: "Not for tons of gold would I exchange the friends I have made in American medicine and throughout the medical world."

He is survived by his widow, Mrs. Margaret Cavanaugh Mosby, a brother, William C. Mosby, a daughter, Mrs. Highland Mary Wood, and a son, John C. Mosby.

Admired by his associates, respected by his colleagues, and loved by his intimate friends and relatives, Dr. Mosby will always remain in the minds of those who worked with and knew him well as a diligent, earnest, and sedulous co-worker and a kind, tolerant, and inspiring leader. With his passing the country has lost a loyal and esteemed citizen and American Medicine and Dentistry, an altruistic and progressive literose devotee.

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